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AFFECTIONS OF THE MOUTH AND THROAT ASSOCIATED WITH THE FUSIFORM BACILLUS AND SPIRILLUM OF VINCENT.[1]

By Emil Mayer, M.D.,

of New York,

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In 1896, in an article on hospital gangrene,[2] Vincent, having found a fusiform bacillus and spirochetæ in his cases, stated that the same bacillus and spirillum could be found in certain anginas of an ulcerative type. In 1897, a year later, Bernheim[3] published his researches in thirty cases of angina and stomatitis, in all of which a fusiform bacillus and spirillum were found. In 1898 Vincent reported fourteen further cases.[4] Bernheim's researches were evidently independently made, and some discussion has arisen as to the priority of their discovery as between these two. Tarassiewicz[5] states that he has seen the report of the Children's Hospital of the Duke of Oldenbourg, in St. Petersburg, 1893, by Dr. Rauchfus. The latter presented cases of ulcero-membranous angina with sharpened bacilli and spirilla, and the photographs prove their identity to those of Vincent. According to Tarassiewicz the credit of the priority belongs to Rauchfus.

Be the credit of priority wherever it may, these anginas are referred to by all writers with the name of the bacillus of Vincent attached. Recently, Niclot and Marotte[6] have presented an exhaustive article on

[1] Read before the Section on Laryngology of the New York Academy of Medicine, November 27, 1901.

[2] Annales de l'Institut Pasteur, 1896, p. 488.

[3] Deutsche med. Wochenschr., 1897.

[4] Bull. de la Soc. des Hôpitaux, March 11, 1898.

[5] Russ. Arch. f. Bak. and Path., 1899, p. 412.

[6] Revue de Médecine, April 10, 1901.

this subject, with a reference list of sixty different papers. It is rather remarkable, in view of all this, that no cases of the kind have been thus far recorded in this country.

Were it simply the announcement of the existence of another bacillus, this might well be relegated to the laboratory or receive but casual mention as an existing fact; one of the interesting novelties found.

The difficulties in the way of diagnosis in membranous and ulcerative lesions of the mouth, however, and the grave importance of the need or not of subsequent treatment, bring the matter urgently before us and make it of vital interest.

My own experience relates to but a single case, but it presents the subject for careful study.

Male, aged twenty-three years, called to see me on September 28, 1901, with the statement that he had had a sore-throat for the past two weeks, which, while not interfering with his business, was daily getting

FIG. 1.

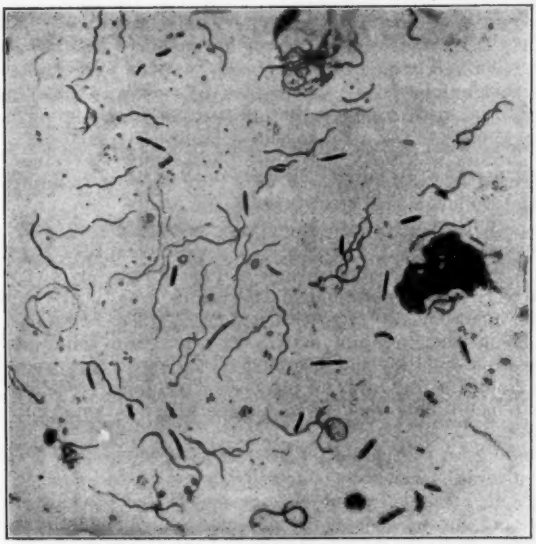
Dr. Mayer's case of ulcero-membranous angina, with the fusiform bacillus and spirillum of Vincent, showing the tonsillar and palatal appearance.

worse. He had seen his family physician, who referred him to me. He was more or less subject to sore-throat, which always yielded to gargles of chlorate of potash. His health had always been good, except for scarlet fever, measles, and pertussis in childhood. His father had died at the age of sixty-eight of pneumonia, a sister at the age of seventeen of the same illness, and two young children, a brother and a sister, in infancy. His mother and a brother are living, and the family in all the collateral branches are healthy. His habits are excellent; he belongs to the higher walks of life; he is a moderate smoker; is tall, athletic, and of fine physique, and has never had any venereal disease, nor has he been exposed to any infected person so far as he knows.

The pain is of a rather mild type, salivation is present, and glands at the angle of the jaw are slightly swollen. He prefers to swallow soft foods; the effort to swallow solids is too painful to persist in so doing.

Examination. Right tonsil is the seat of three separate spots of white deposits, irregular in shape, reddened about the edges, easily detached, having an ulcerated and bleeding surface underneath. Between the membranous deposits is normal tonsillar tissue. Two similar deposits exist on the left tonsil. Although the membrane was readily detached, crumbling even when touched, it re-formed very promptly. A few days later a few spots showed to the left of the uvula, and subsequently

FIG. 2.



Showing the fusiform bacillus and spirillum of Vincent from Dr. Mayer's case.

all the spots joined into a single mass of a pearly white color, and there was a line of deposit over the gums of both upper and lower teeth.

A specimen had been removed for microscopical examination and the bacillus and spirillum of Vincent found. In order that the bacteriological examination should be free from possible extraneous additions, the patient was sent to the laboratory, where spreads were made by the bacteriologist, whose report is here appended.

THE MOUNT SINAI HOSPITAL, PATHOLOGICAL DEPARTMENT,
NEW YORK, November 7, 1901.

DEAR DR. MAYER: The results of the examinations in your case are as follows:

1. The small piece cut from the tonsil on September 30th showed areas of necrosis and areas of inflammation, with suppuration. In some

parts there were masses of fusiform bacilli, apparently the same later found in spreads from the affected areas. The areas of necrosis were quite granular in appearance, there being an occasional small, round nucleus present.

2. The spreads made by myself from scrapings of the surface of the ulcerations showed the same appearance on both days on which they were made (October 4th and 5th).

Of cellular elements there were present mainly polymorphonuclear neutrophilic leucocytes and a few small mononuclear cells, with a round, comparatively large nucleus and no granulations.

In the spreads were found numerous bacilli and spirochetæ, and very few short chains of streptococci. The bacilli varied in length from 4 to 12μ. They were non-motile. Some were completely decolorized by the Gram procedure, some partly, and some not at all. The bacilli were fairly thick; some were thicker at one pole than at the other; many were curved, some quite irregularly. Most were pointed at both ends; a fair number were swollen in the centre. They occurred as diplobacilli, in small masses or lying at angles to each other in groups, somewhat similar to the arrangement of diphtheria bacilli.

They stained well with the usual basic stains, but were best seen when diluted carbol-fuchsin was used. Often clear places were seen in the bacilli, but no spores could be found.

The spirochetæ were rather long and were distributed irregularly. They were non-motile. They were decolorized completely by the Gram procedure. Staining reactions same as for the bacillus.

Cultures from the secretions of the ulcers in ordinary media, aërobically and anaërobically, showed streptococci only. Inoculations were also made on glucose-serum media, both fluid and solid,[1] and on the ordinary serum-bouillon and serum-agar. They were grown aërobically and anaërobically. Only streptococci were found. There was no increase in bacilli or spirochetæ in the condensation water in the serum-agar tubes.

The findings in this case are the same as those made in a number of cases of ulceration in various parts of the mouth by Vincent and later by Bernheim. The only difference present is that the bacteriæ in your case were not motile, whereas the spirochetæ have generally been found quite motile. Reports as to the motility of the bacilli vary. The picture, however, is so characteristic that the difference is of no moment.

As to the culture experiments: Niclot and Marotte[2] used serum-media of the same kinds that I did (none, however, with glucose). They succeeded in some of their cases only in obtaining results either in fluid media or in the condensation water of the solid media. They never obtained pure cultures; streptococci were always present.

I made a few tests with the 1 per cent. acetic-acid bouillon recommended by Silberschmidt,[3] but all the tubes remained sterile.

The time of observation of all culture tubes was four days.

Very truly yours,

E. LIBMAN.

The treatment instituted was frequent gargles of boric-acid solution and daily application of tr. iodin. comp. For nearly two weeks the

[1] See Libman, Journal of Medical Research, vol. i. p. 84.

[2] Revue de Médecine, 1901, No. 4.

[3] Centralblatt für Bakteriologie, Band XXX., No. 4.

deposit seemed to remain, when it gradually began to disappear, and on October 25th, four weeks after I first saw him, there was no sign of any deposit, the patient felt well and has remained so.

There is a varied nomenclature regarding this affection. Vincent himself and subsequently Freyche[1] use the term diphtheroid angina in mentioning the condition. Panoff[2] and Marian[3] speak of it as chancriform angina. Bonnus and Deguy[4] call it ulcero-membranous angina; Athanasiu,[5] merely ulcerous angina; while Salomon,[6] in a recent article, defies the translator and, in the happy manner of word-building so potent and so beautiful in the German language, calls it "spirochäten-bazillenangina."

This affection seems to have a predilection for the adult male, and it is said to be due to the use of tobacco, eruption of a wisdom tooth, defective teeth or those covered with tartar, lymphoid tendency, syphilis, and mercurial stomatitis. It is mainly an affection of the tonsils, but also occurs as a stomatitis. It is generally conceded that the presence of the bacillus of Vincent precludes the existence of diphtheria bacillus, de Stoecklin[7] having pretty well established that fact. Because of our inability to inoculate with or to produce pure cultures of these bacilli and spirochetæ, we are not prepared to say that these are the sole controlling factors in the etiology of this affection, hence the title of this article reads "associated with" rather than "caused by" them.

These bacilli may readily be associated with or engrafted upon a syphilitic soil, Salomon[8] having recorded two cases in which the bacillus and spirillum were found. After a time the characteristic deposits ceased, and with it the microscopical findings and undoubted mucous patches appeared.

The symptoms present are of the slightest kind, as a rule, the deposit existing for quite a time without being noticed. The lesion has a distinctly chancriform appearance at first, and, while usually situated on the centre of the tonsil, it is often on its most dependent portion. The deposit is white, soft on top, readily detachable, and leaving an excavated and bloody surface. There is a certain amount of induration, the breath has a peculiar fetid odor, salivation is present, submaxillary glands are infiltrated. There is slight pain in deglutition. In a case recorded by C. Nicolle[9] there was an ulceration on the lip similar to those on the tonsils, and on the upper part of the face there were several œdematous spots, painful to the touch, with erythema and

[1] Thèse de Toulouse, 1899.
[2] Thèse de Nancy, 1899.
[3] Nord. Medical, March 1, 1900.
[4] Journal des Praticiens, May 19, 1900.
[5] Soc. de Ped., June 12, 1900.
[6] Deutsche med. Wochenschr., No. 34, August, 1901.
[7] Centralblatt f. Bakteriologie, 1898, No. 17.
[8] Loc. cit.
[9] Normandie Med., June 1, 1899.

discoloration of the skin. Niclot and Marotte[1] mention a complication of appendicitis in one and pseudo-rheumatism in another.

The borders of the gums, the lips, and tongue are the principal seats of this affection in the mouth. The duration of the affection seems to be from eight days to four or six weeks. The prognosis is, as a rule, very good, but there is a tendency to recurrence. Like all other infectious maladies, it is susceptible of complications of a various and sometimes serious nature. One fatal case is recorded where there was an ulcero-membranous stomatitis complicated by infectious purpura. Endocarditis may follow the condition.

The treatment consists in the use of boric-acid solution as a gargle, the local application of iodine and of peroxide of hydrogen.

Of utmost importance is the question of diagnosis, the two most important conditions differentiated being diphtheria and syphilis. In the former the exposure to contagion, the presence of temperature, cardiac depression and albuminuria will serve to differentiate, but in both the bacteriological diagnosis must be resorted to as definitely settling the matter.

Clinically, this affection is exceedingly difficult to differentiate from any one of the forms of specific disease, and hence the importance of its careful study. This becomes necessary not only for the patient's future welfare, but also for his present needs, for it is a well-known fact that mercury in any form interferes with the healing of an ordinary stomatitis.

It must also be borne in mind that syphilis may be present, and we may have a membranous deposit with the fusiform bacillus and spirillum associated with an underlying syphilis, and here the question arises as to what treatment should be instituted. To this I would answer, treat the angina first, and eliminate that, incidentally, if necessary, giving iodide of potassium internally until the angina is entirely healed.

In differentiating from a primary lesion or tonsillar chancre, the question of induration can hardly help us, for the reason that palpation in this region is almost a physical impossibility. The presence of cervical glands in syphilis may be of some help, as will also the history of exposure.

The duration of both may be long. In the secondary conditions the ulceration is much more apt to be deep and continuous. In the tertiary form gummatous lesions are more apt to be prevalent, and the history by this time has probably been sufficiently clear; but, under all circumstances, the final decision as to the presence of these bacilli and spirilla must depend upon the results of the bacteriological examination. Without the laboratory many an unfortunate would be

[1] Loc. cit.

promptly placed upon anti-syphilitic treatment, doomed to take quantities of drugs for years and years, fearful lest at any time there might be an outbreak of a disease he never had; if unmarried, always doubting his right to take such a step, and if married, shunning those near and dear to him.

It is surely worth our while to make haste slowly, and to examine every single case of membranous sore-throat with all the means at our command, or, if not every case, surely those whose history is not clear and where there may be the least doubt.

[The gross illustration is made from a sketch kindly drawn by Dr. Percy Fridenberg. The photomicrograph (Fig. 2) shows the bacilli and spirilla very clearly.]

A CASE OF ACUTE CHOLECYSTITIS WITH GANGRENE; CHOLECYSTECTOMY; RECOVERY.[1]

By Francis D. Donoghue, M.D.,
INSTRUCTOR IN OPERATIVE SURGERY, TUFTS COLLEGE MEDICAL SCHOOL.

The report of the following case of cholecystitis with gangrene is of interest as showing the rapidity with which a case of quiescent cholelithiasis may be converted into an acute fulminating condition, requiring the prompt operative treatment which should be given to all cases of severe perforative peritonitis, and goes to prove the statement "that cholelithiasis is a disease which becomes dangerous through cholecystitis or its consequences."

Julia E., residing in Salisbury, Mass., aged fifty years, married, and the mother of six children, was taken with a severe pain in the region of the gall-bladder on Sunday, May 5, 1901. It began about 11.30 P. M., waking her from a sound sleep. Pain lasted for twelve hours. She vomited during the night, and the vomiting continued until 3 P. M. Monday. Monday night she felt so much easier that she went home on the electric car, a distance of three miles. Tuesday she felt sick, but there was no vomiting or severe pain. There was a good deal of tenderness over the upper abdomen, and a bunch was discovered under the ribs. Tuesday evening she called Dr. Spaulding, who found her with a temperature of 102.5° F., a pulse of 100, and on palpation discovered an elongated tumor extending below the edge of the liver, very sensitive on pressure. The next morning she was taken to the Anna Jacques Hospital in Newburyport.

On Wednesday P. M., May 8th, I saw her in consultation with Dr. Spaulding and Dr. Abby Noyes Little. Found a fairly stout, strong, well-nourished woman, giving the following history: Had usual dis-

[1] Read before the Suffolk District Medical Society, Surgical Section, December 4, 1901.

eases of childhood, including scarlet fever and diphtheria. As a child was subject to sick headaches. After the age of thirteen years was never seriously ill until last fall, when she had an attack of "malarial fever," as she called it. Had chills and fever for six weeks, but there was no pain or stomach or bowel disturbance. "Years ago" would occasionally have sudden severe attacks of pain across the upper abdomen. They lasted from three hours to half a day, and were relieved by vomiting. Never was jaundiced and never called a physician. She has been free from such attacks for the past ten years. Six or seven years ago had a "nervous attack" lasting eight weeks. For three or four years has had by spells a sense of pressure and some distress in region of gall-bladder, especially after exertion. Menopause a year ago, accompanied by indefinite nervous symptoms. Examination of abdomen showed in right hypochondrium an elongated tumor extending about three fingers' breadth below outer border of ribs and lying under the right rectus muscle. It was dull on percussion, sensitive on pressure, and moved slightly with respiration. The remainder of the abdomen, as well as the heart, lungs, and urine, presented nothing abnormal. Stool following enema was of fairly normal color and formed. Temperature was 103.5° F.; pulse, 130. There was no icterus of the skin or of the sclera, and the liver did not appear to be enlarged.

Operation at 5 P. M., under ether, Dr. Little assisting. The incision through the outer border of the right rectus muscle opened directly onto the gall-bladder, which was found tense, distended, and apparently full of calculi. Its surface was smooth except for some fibrin upon the inner end, and there were no adhesions. At the outer end was a very thin and apparently necrotic spot. The bile-ducts were palpated, and no obstruction other than in the cystic duct could be made out. The gall-bladder was freed from its "liver bed," the duct below the obstruction was clamped, a strong silk ligature was tied, and the gall-bladder cut between the clamp and tie and removed. Two gauze drains were passed down to the stump and the incision closed by interrupted catgut sutures of rectus fascia, muscle and peritoneum, except at upper angle, out of which the gauze drain and end of silk ligature were brought. Skin closed by interrupted silkworm-gut sutures.

For the subsequent history I am indebted to Dr. Little. Bowels moved on the second day. First dressing on fourth day; wound clean; no discharge. Sat up on the fourteenth day. Ligature came away on the sixteenth, and she was discharged on the twenty-third day, with the wound entirely closed. On the tenth and eleventh days the temperature ran up to 101° F., accompanying an attack of facial neuralgia. There was no trouble in the wound.

Dr. Spaulding writes me under date of December 2d as follows: Mrs. E. is in perfect health, and looks it. She is at work nursing; eats anything and everything, and suffers no distress.

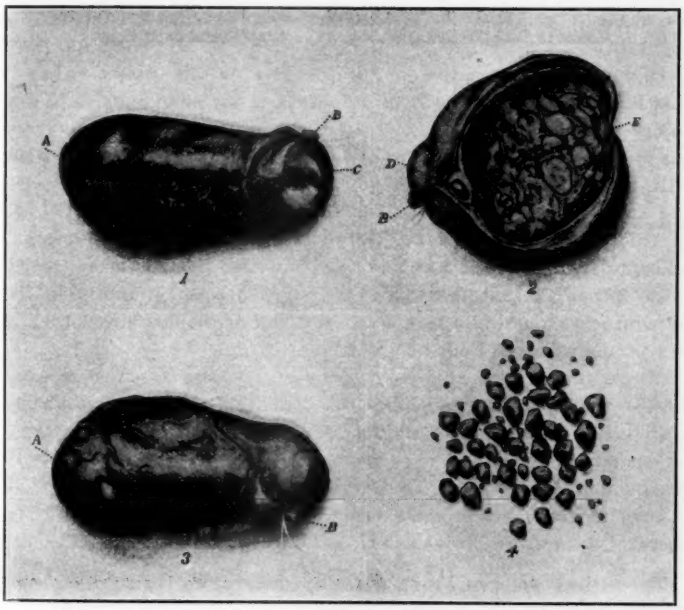
Report of Dr. W. F. Whitney on Specimens.

Gall-bladder distended to twice natural size. Walls hemorrhagic and dark red in color externally. Internally, mucous membrane deeply ulcerated in places, as if from pressure. Where this is not the case it is intensely injected. Summit (or apex) of gall-bladder shows a dark green discoloration, as if gangrenous. Cystic duct blocked by

small calculus. Contents of gall-bladder: mucopurulent secretion and large number of calculi.

Diagnosis. Acute cholecystitis, with beginning gangrene.

Cholecystectomy in this case seemed to be the operation of choice, as it should be in most cases where there is gangrene or necrosis of the mucous membrane or of the gall-bladder wall. It removes immediately the major part of the infection and does away with the possibility of a recurrence, and obviates the possibility that even after a successful cholecystotomy distress may again occur because of extensive adhesive processes.



The absence of icterus, clay-colored stools, or stones in the feces was noted in this case, and in this connection the conclusions of Professor Kehr, of Halberstadt, from an analysis of 535 laparotomies for gall-stone disease, are interesting. He believes "that icterus is wanting almost always in an inflammatory process of the gall-bladder and in the beginning of gallstone disease. The passage of the stone in the feces is not so common as it has hitherto been considered, for most colics are unsuccessful; that is, the stone remains in the gall-bladder. Finally, it is time that the physician should give up the view that icterus belongs to cholelithiasis, as in 80 per cent. of all gallstone cases it is absent."

PHRENIC NERVE INJURIES. REPORT OF A CASE. ANATOMICAL AND EXPERIMENTAL RESEARCHES, AND CRITICAL REVIEW OF THE LITERATURE.*

By W. E. Schroeder, M.D.,
ASSOCIATE PROFESSOR OF SURGERY, NORTHWESTERN UNIVERSITY MEDICAL SCHOOL; SURGEON TO THE WESLEY, PROVIDENT, AND COOK COUNTY HOSPITALS.

WITH

ANATOMICAL OBSERVATIONS ON THE INNERVATION OF THE DIAPHRAGM.

By F. R. Green, A.M., M.D.,
ASSISTANT DEMONSTRATOR OF ANATOMY, NORTHWESTERN UNIVERSITY.

In a careful examination of the literature on this subject, one finds that most authorities agree that a division of the phrenic nerve or any injury to it is fatal, or certainly very dangerous. I was led to doubt the accuracy of this conclusion, first, by a case of paralysis of the right half of the diaphragm, complicated by a diaphragmatic hernia following an anterior poliomyelitis which I saw in 1892; secondly, by Dr. Murphy's[1] nitrogen gas method of collapsing one lung for tuberculosis; and thirdly, by cases of recovery from surgical pneumothorax, which made me think that the worst that could happen in injury to the phrenic nerve would be a paralysis of one-half of the diaphragm leading to a partial collapse of one lung only.

My skepticism led me to take liberties with the nerve, so that recently I have pinched it systematically every time it has been exposed during a tubercular gland operation, after Fenger's[2] method, and when, during an operation for a tumor of the neck, it was found impossible to remove it without dividing the nerve, I did so unhesitatingly.

I am indebted to Dr. J. Eskridge for this interesting case. It was operated upon at the Provident Hospital, August 30, 1900.

The patient was a widower, white, aged sixty-two years; American, an upholsterer by occupation.

The family history was good, with the exception that two cousins on the mother's side died of cancer of the uterus, and two sisters of heart disease. Father died of pneumonia at the age of eighty-seven.

Patient had been a heavy whiskey drinker until nine years ago, and he still smokes and chews a great deal. He had the usual diseases of childhood, but no others. Twelve years ago his lower jaw was fractured in a railway accident, and eight years ago he was cut in the right supraorbital region by an ax, which resulted in a paresis of the orbicularis palpebrarum muscle. There were no congenital deformities.

The present trouble began nine years ago, when the patient noticed a tumor on the left side of the neck about as large as a hazelnut, but painless. This increased in size very slowly during the first four years,

* Read before the Chicago Medical Society, March 18, 1901.

at the end of which time it was about as large as a walnut and painful upon pressure. During the last five years the tumor has grown somewhat more rapidly and has become more painful, and the pain has been constant during the last year.

The tumor is situated on the left side of the neck immediately behind the sternocleidomastoid muscle and partly covered by it. (See Fig. 1.) It begins three-fourths of an inch above the clavicle and extends upward two and one-half inches. It is two and one-half inches long in the long diameter, which is parallel with the long axis of the body; its inner margin lies one inch from the left border of the trachea. The skin covering the tumor is normal and freely movable. There are no large vessels to be seen. The tumor feels hard, and is so

FIG. 1.

Before operation.

firmly attached deep down that it cannot be moved. No fluctuation, pulsation, or bruit. No enlarged glands. Aspiration shows, macroscopically, bright red blood in considerable quantity; microscopically, nothing but blood-corpuscles, neither epithelial nor other cells being found. No pressure symptoms.

The physical examination of the patient resulted as follows:

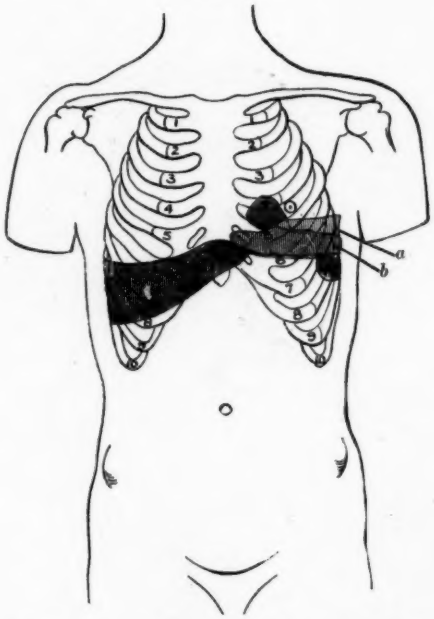
Body well nourished; weight, 160 pounds; no recent loss in weight; no abnormality of skin or glandular system. Pulse, temperature, and respiration normal; appetite good; walls of arteries soft; no symptoms of pressure on sympathetic nerves. Paresis of orbicularis palpebrarum due to old injury. Heart and lungs normal. Special attention was paid to the lungs and the position of the diaphragm. Liver,

spleen, etc., normal. Urine and blood count normal. Patient was prepared for operation by usual method.

An incision was made from the posterior edge of sternocleidomastoid muscle one inch below the mastoid process to the clavicle. The external jugular vein was ligated and divided between the ligatures. Continuing the dissection, the sternocleidomastoid muscle was liberated and the spinal accessory nerve found and dissected out to the trapezius muscle.

The tumor came into view and showed a capsule which was not adherent to any great extent to the surrounding tissues, but was firmly

FIG. 2.*



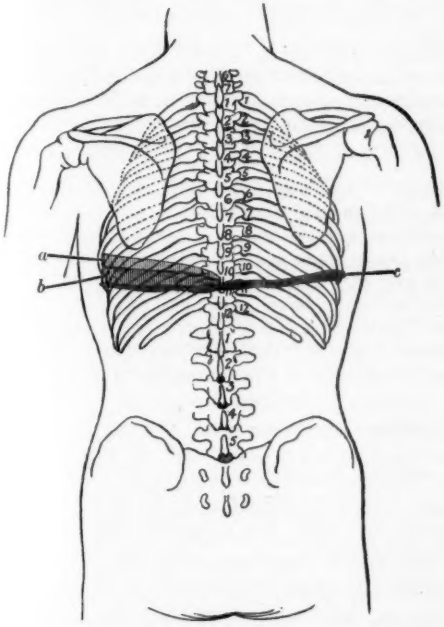
Anterior view. *a*. Relative dulness. *b*. Absolute dulness.

attached deep down. It was at once noticed that the phrenic nerve lay over the upper outer part of it and that its lower branch was but slightly visible. The nerve was quite tense, but responded twice when it was pinched. As I believed the tumor to be of a malignant nature, I did not wish to divide it for fear of implanting cells in the wound. Therefore, I tried to dissect the nerve from the tumor, but in doing so the small branch from the fifth cervical ruptured, and soon after the main branch from the fourth cervical did likewise.

* Figs. 2, 3, and 4: Findings upon percussion after division of nerve. *a*. Relative dulness, *b*. Absolute dulness.

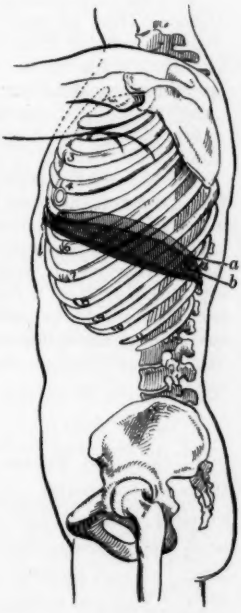
I noticed no material change in the patient excepting an increase in the number of respirations to thirty-two. The tumor was so situated that the upper and main root of the phrenic traversed the upper outer part of it, while the lower root came from underneath. It was firmly attached to the foramen formed by the third and fourth cervical vertebræ, and its removal caused a venous hemorrhage from this point. The fat and various glands were removed from the side of the neck. I followed the phrenic down to the clavicle, injuring the thyroid axis, which produced some hemorrhage. No other branches of the phrenic were found. In trying to unite the divided parts of the nerve, it was

FIG. 3.



Posterior view. *a*. Relative dulness. *b*. Absolute dulness. *c*. Normal side.

FIG. 4.



Lateral view. *a*. Relative dulness. *b*. Absolute dulness.

impossible to find the little branch from the fifth cervical, but the ragged end of the trunk and that of the fourth root were cut off in order to give uninjured ends, and reunited by means of silk sutures. The nerve was now pinched below the suture, and responded. There was no cough, no singultus, no sneezing, none of the symptoms reported in other cases. The wound was closed in the usual manner. After the patient recovered consciousness, I asked him whether he had any difficulty in breathing or any pain in the region of the diaphragm, but he said he had not. However, the respirations remained more frequent (24 to 30) for four or five days, after which they came down to

20, and remained so. About eight hours after the operation, percussion showed that the left half of the diaphragm was two and one-half inches above its normal position. An area of the lower portion of the lung, an inch and a half in width, showed relative dulness. (See Figs. 2, 3, and 4.) Litten's sign was absent.

Auscultation over dull area showed vesicular breathing rather distant. Slight abdominal retraction on inspiration. During his three weeks' stay in the hospital the position of the diaphragm remained the same, but the area of relative dulness gradually disappeared and the lung returned to its normal condition. Measurement of the thorax showed right half 44 cm. Deep expiration, 41½ cm. Inspiration, 44½. Left half, 43 cm. Deep expiration, 41½ cm. Inspiration, 43½.

At present the patient is employed as usual, and is suffering no inconvenience from his paralysis.

Macroscopical Examination. The tumor is solid but very vascular. It is two inches in its long diameter and an inch and three-fourths in its transverse diameter. Upon cutting the tumor in half the interior presented a solid consistency, but many large vessels were to be seen. A few small areas, 1 cm. square, were somewhat softer than the main body of the tumor.

Microscopical Examination. One sees numerous connective tissue fibres around the periphery, forming a well-defined capsule, and many areas of leucocytes around which there seems to be young connective tissue. In the centre of the tumor there is a hemorrhage into the tissue. This is very recent, and is probably due to the puncture made by the aspiratory syringe. There are many large and small bloodvessels, but no new ones to be seen. Leucocytes are grouped at various places, resembling an inflammatory process. I believe the tumor to be a fibroma and not, as was thought from the clinical evidence, a fibro-sarcoma.

Experimental Researches.

1. *Upon the Human Subject.* As before stated, in all recent operations for tubercular glands, I have searched for the phrenic nerve and pinched it below the lower root, with the following results: Each time the corresponding side of the diaphragm contracted, producing a sudden and decided abdominal rising immediately below the costal arch. This has been done in eighteen cases, ten right and eight left. In two cases, one right and one left, there was some pain in the region of the corresponding half of the diaphragm, which subsided in each instance before the end of forty-eight hours. The symptoms commonly ascribed to irritation of the phrenic, *i. e.*, sneezing, coughing, and hiccoughing, were not observed in a *single instance.*

2. *Upon Dogs.* The animal experiments have been made upon dogs only, because they resemble man in their type of respiration, which is costo-abdominal. The method of conducting the experiments has been as follows: The circumference of the thorax was taken at the tip of the ensiform cartilage, and again from one to two and one-half inches above it. The number of respirations per minute were recorded

before and after division of the nerves, and the change in type of respiration noted. Ether was the anæsthetic chosen. The usual antiseptic precautions were observed. After exposing the nerve the interrupted electrical current was applied, and then the nerve was pinched with an artery forceps. The nerve was now either evulsed or resected. Later in this work I weighed the animals before the operation and each week thereafter.

Post-mortems were conducted from one week to four months after the division of nerves. For this purpose in most cases the dogs were deeply anæsthetized with chloroform, the thorax opened, the nerves found and stimulated with the electrical current. The various phenomena were observed, and specimens and drawings made. For the details of these experiments see Table I.

It may not be amiss to state here that before obtaining the results shown in this table, there were eight animals that died, but all of these can be accounted for without detracting in the slightest way from the above results. At first I evulsed the nerve, tearing out from three to five inches of it. Out of four cases only two survived. (See Table I., Cases 1 and 2.) Post-mortems of the two that died showed in one case of double evulsion a double pneumothorax and in the other a pneumopyothorax. In the latter case, I heard the rush of air into the pleural cavity, and, infection occurring, the animal died. Evulsion of the nerve lacerates the pleura, and with an infection is liable to result in death from pneumopyothorax; therefore, I abandoned this method of removing the nerve and resorted to resection outside of the pleura, removing as much of the nerve as was possible in each case.

A third animal died from the anæsthetic before any incision had been made.

Five died as a result of experiments with chloretone as an anæsthetic. These animals were given two-tenths of a gramme per kilo, and operations were begun two hours later. They were not completely under, so they were given two-tenths more per kilo, and operations begun after another hour. By this time they were profoundly anæsthetized, but none of them recovered consciousness. They slept from two to four days, when they died.

Weight. The first seven were not weighed, but my observations would lead me to state that the first week or two they lost in weight, after which they gained and again came back to their normal condition. In the last three it will be seen that they lost in weight for the first week, but came back to normal or nearly so. The loss was not great in any instance.

Measurements. It will be seen that in the single division there was an increased thoracic expansion and usually a slight abdominal retraction, which were more evident on the divided side than on the normal side.

TABLE I.

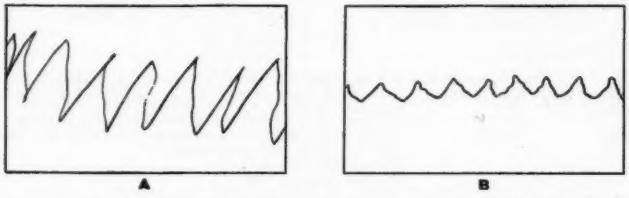
No.	Date of operation. 1900	Sex, size, and weight.	Measurements in inches. Before operation. At ensiform. Exp.	At ensiform. Insp.	Above. Exp.	Above. Insp.	After operation. At ensiform. Exp.	At ensiform. Insp.	Above. Exp.	Above. Insp.	Respiration: frequency and type. Before division.	After division.	Stimulation of nerve before division. Electrical current.	Pinching.
1	Oct. 24	F. small,	15	15 1/8	15 3/8	16	15	15 3/8	15 3/8	16 1/4	All dogs costo-abdominal type. 24	Costal, abdominal retraction slight. 35	Tetanic contract'n.	Short well-defined contract'ns
2	Nov. 5	M. large,	25 1/4	26	26 1/4	27 1/8	25 3/4	26 1/2	26 1/4	27	19	Pronounced costal, inverted. 30	Tetanic contract'n.	Short well-defined contract'ns
3	Nov. 24	M. medi'm	21	21 1/4	22	22 1/8	21	21 1/4	22	22 1/2	23	Increased costal, abdomen almost passive; no pronounced retract'n. 27	Tetanic contract'n.	Short well-defined contract'ns
4	Dec. 3	F. small,	14	14 1/8	15	15 1/4	14	14 1/4	15	15 1/8	26	Decided & somewhat labored costal; inverted. 36	Tetanic contract'n.	Short well-defined contract'ns
5	Dec. 3	M. small,	21	21 1/4	22	22 1/4	21	21 3/8	22	22 1/2	20	Costal, slight retraction only. 24	Tetanic contract'n.	Short well-defined contract'ns
6	Dec. 7	M. small,	20	20 1/4	20 1/2	20 3/4	20	20 1/2	20 1/2	21	Costal, slight retraction only. 27	Costal, slight retraction only. 24	Tetanic contract'n; contr. of left leg also.	Short well-defined contract'ns
7	1901 Jan. 23	M. large, 50 lbs. Feb. 12 51 lbs. Feb. 19 51 lbs.	30	30 3/8	...	...	30 1/2	31 1/2	...	...	25	30	Tetanic contract'n.	Short well-defined contract'ns
8	Jan. 23	M. small, 12 lbs. Feb. 12 10 lbs. Feb. 19 11 lbs.	13 1/8	13 1/4	...	...	13 1/8	13 3/8	...	...	44	Costal, some abdominal retract'n. 40	Tetanic contract'n.	Short well-defined contract'ns
9	Jan. 23	M. medi'm 25 lbs. Feb. 12 24 lbs. Feb. 19 24 lbs.	19	19 1/8	...	...	19	19 1/4	...	...	24	Breathing only slightly altered. 26	Not pronounced	Not pronounced
10	Jan. 23	M. small, 15 lbs. Feb. 12 12 lbs. Feb. 19 15 1/4 lbs.	15	15 1/8	...	...	15	15 1/4	...	...	30	Costal, but not pronounced. 32	Tetanic contract'n.	Short contract'ns

TABLE I.—*Continued.*

Division of nerve: right, left, both.	Electrical stimulation before death.	Preliminary to post-mortem.	Postmortem findings. Remarks.
Left, evulsion 5 inches.	Tetanic contraction of right, none of left.	Nov. 14, etherized; an incision was made below ensiform through abdominal wall revealing active contraction of r. half of diaph. and passive of left half; kymographic tracing was taken. See Fig. 5.	Nov. 14. Some atrophy of left half of diaph. crossing the median line; no regeneration of left nerve. Dog was six weeks pregnant; uterus contained five embryos; well nourished.
Both, evulsion right 3 in. left 3¼ "	April 16. Chloroform was given, thorax was opened, and left nerve found and stimulated; left half of diaphragm contracted slightly, showing partial regeneration; no reaction of right nerve.	Inverted type of respiration.	Marked atrophy of diaphragm, excepting at costal margin; partial arrest of development of abdomen and its contents; no hernia of diaph. Exercised the dog violently Dec. 28, 1900, and Jan. 15, 1901; he did not seem to suffer from labored respiration; still inverted type of respiration; he had been infected.
Right, resection 1 inch.	Jan. 17, 1901. Dog was shot and current applied immediately; no reaction of right nerve; normal contraction of left side.	Just before killing the dog, measurements at ensiform on expiration, 21 in.; on inspiration, 21⅜ in.; 2⅝ in. above ensiform, on expiration, 22 in.; on inspiration, 22⅜ in.	Atrophy of right half of diaphragm, which was thin, yellow, semi-transparent; a normal zone ¼ inch wide was formed at the periphery; right nerve had regenerated, but the right half of diaphragm did not respond to stimulation.
Both, resection 1½ inch.	No reaction.	....	Dec. 17. Both nerves show some proximal regeneration: atrophy of diaphragm not very decided, because only 14 days passed between the division and post-mortem.
Right, resection 1½ inch.	No reaction; left normal.	Measurements before killing animal: ensiform, expiration, 21 in.; inspiration, 21⅜ in.	Dec. 15. Some atrophy of diaphragm.
Left, resection 1½ inch.	Left reaction; right reaction.	Measurements at ensiform, expiration, 21¼ in.; inspiration, 21 in.	Dec. 14, *i. e.*, 7 days after operation, slight atrophy of left half of diaphragm only.
Both, resection 1½ inch.	No reaction on either side.	.....	Feb. 26, 1901. Very decided atrophy of both sides; right side, costal margin, one finds an area which is not atrophic, because it receives a nerve from intercostals. Fig. 6. Costal margin not atrophied.
Left, resection 1 inch.	No reaction of left; reaction of right.	.....	Feb. 19, 1901. Atrophy of left half of diaphragm; none of right. Fig. 7.
Both, upper two roots found only.	Reaction of both sides in thorax.	.....	March 9, 1901. V-shaped portion of diaphragm at ensiform cartilage is atrophied, while the remaining portion is normal. Fig. 8.
Right, resection 1 inch.	Reaction in left; none in right.	.....	Mar. 9, 1901. Atrophy of right half of diaphragm, excepting one centimetre of costal margin.

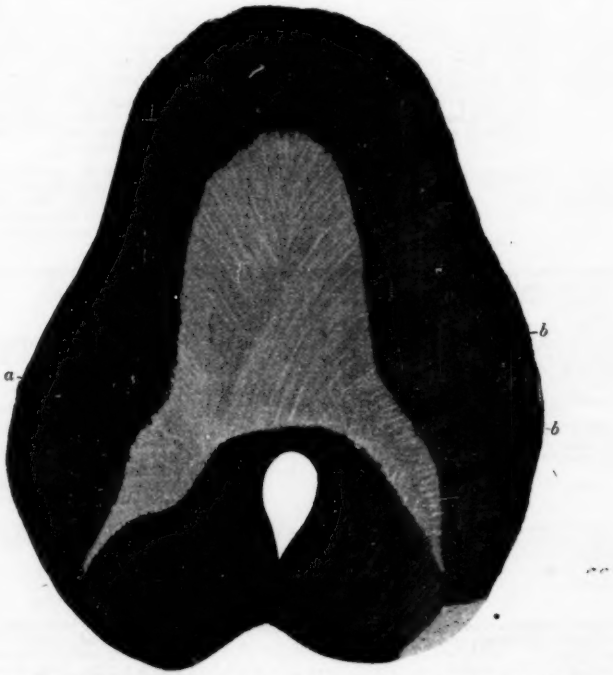
In the double divided cases there occurred an inverted type of respiration, *i. e.*, decided abdominal retraction on inspiration, and increased thoracic expansion due to action of accessory respiratory muscles.

FIG. 5.



a. Excursion of right half of diaphragm. *b*. Passive movement of left or paralyzed half.

FIG. 6.



Resection of both phrenic nerves. Dog No. 7. *a*. Normal area. *b*. Non-atrophied margin.

In Case 9, as the upper branches *only* were divided, the animal did not suffer much of a change because the lower branches supply by far the greater part of the diaphragm. (See Fig. 8.)

Electrical Stimulation. In all cases I used the induced current after the Du Bois Raymond method,[3] always with a positive result, *i. e.*, tetanic contraction before division and no reaction after the fourteenth day. In Case 6 the animal was killed only seven days after division, and on stimulation there was a slow, feeble contraction, showing either that the nerve was not yet entirely degenerated or that the atrophy of the diaphragm was not yet complete. In Case 9 the current produced a contraction of diaphragm supplied by lower root. (See Fig. 8, *b b*.)

Excursions of Diaphragm. In dog 1, I opened the abdomen in the median line immediately below the ensiform cartilage and inserted a

FIG. 7.

Resection of left phrenic nerve. Dog No. 8. *a*. Non-atrophied margin. *b*. Atrophic area. *c*. Œsophagus.

glass rod, pushing it against first one side of the diaphragm and then the other, taking a kymographic tracing (see Fig. 5), with the result that the right half contracted normally, producing a rise one-half inch in height on inspiration and corresponding depression on expiration. The left half produced a very small rise, one-eighth inch, representing the influence of the contractions of the right side. There was no contraction of the left half. In other animals, I observed the same phenomena, *i. e.*, that the paralyzed side was passive and only acted upon by the contractions of the normal side.

Symptoms. The diaphragm becomes relaxed after the division of the nerve and arches up into the thorax. The type of respiration becomes

increased costal when one nerve only is divided, and inverted when both nerves are divided. The accessory respiratory muscles become very active.

There was no sneezing or coughing in any of the animals. In one case of double division the respiration became labored, but it remained so for two or three days only.

The Post-mortem Findings. Macroscopical. In the cases where the dog was killed from seven to fourteen days after division the atrophy

FIG. 8.

Resection of upper roots. Dog No. 9. *a*. V-shaped atrophic area. *b, b*. Normal area. Crura.

was not very great and the color was reddish-yellow. In cases where a long time had elapsed between the operation and the post-mortem, the atrophy was very decided, the paralyzed part was thin and flabby, the diaphragm was pale yellow, and in some of the older cases translucent. In all cases there remained a margin from one-quarter to three-eighths of an inch wide at the costal border, which retained its normal color and thickness. This margin is probably supplied from the intercostal. (See Figs. 2, 3, and 4.) Again, in the specimen taken from dog 7 (Fig. 6, *a*), it will be seen that there remains a relatively

normal area on the right side, which was probably supplied by a long branch from the intercostal nerve. In all three figures there is a normal area at the crura. These areas evidently receive an independent nerve supply.

In Fig. 8, where the root coming from the upper branch has been divided (dog No. 9), there is a V-shaped area with its apex toward the sternum extending a distance of two and three-quarter inches on each side toward the spine. This is evidently supplied by the upper branches, the lower branch supplying nearly all of the remaining portion of the diaphragm. In dog No. 3, although the nerve had regenerated after fifty-four days, there was no electrical reaction because of atrophy of the diaphragm.

There is very little available literature upon the experimental work on this subject. Sir Astley Cooper[4] reports having experimented by putting a ligature on the phrenic nerve of a rabbit. The animal suffered from labored respiration and died twenty minutes later. Post-mortem showed that the nerve had been cut.

Henocque and Eloy[5] reported some experiments in 1883.

Billard and Cavalie,[6] in 1898, reported on the sequelæ of section of both phrenics in the dog.

Review of Literature. The literature on this subject is very meagre and confusing, and the conclusions reached by various writers are often diametrically opposite. Thus Gross[7] says: "The division of the phrenic nerve is nearly instantaneously mortal."

Erichsen,[8] in his eighth edition, says: "The division of the respiratory nerves on one side only would, in all probability, be fatal in man by interfering with the proper performance of the respiratory act;" but in his tenth edition[9] he modifies his conclusion, and says: "Division of the phrenic nerve would necessarily, by interfering with respiratory movements, induce a tendency to congestion and inflammation of the lungs; and although such an accident must be a very rare one in cases of ligation of subclavian for axillary aneurism, yet it undoubtedly has occurred as I have myself witnessed in one case."

Ranney[10] says: "Since the phrenic nerve arises in the upper cervical region, any lesion of it will cause respiration to be interfered with and create dyspnœa and hiccough; but respiration will not be arrested, since the pneumogastric nerves continue to excite it, and the auxiliary muscles can expand the chest without the action of the diaphragm. Should the lesion be a surgical one, the respiratory centre of the medulla may be affected and death take place from asphyxia, but I do not think such a result can be explained as a simple effect of paralysis of the phrenic nerves alone."

Hueter-Lossen[11] says: "Paralysis of one-half of the diaphragm puts life in immediate danger. This is evident not only from observation

of injuries to the phrenic itself, which are rare, but from injuries to the roots of the nerves in the spinal cord, which determine practically always a fatal termination. These latter may be produced by injuries from the fourth cervical vertebra down, or by degenerative processes spreading from below upward. They are always indicated by paralysis of this principal muscle of respiration, the accessory muscles having been already more or less paralyzed, probably completely. Diaphragmatic paralysis is always easy to determine, since by it alone is inspiration performed under ordinary circumstances."

Diebel[12] calls attention "to the property possessed by nerves and vessels of resisting the encroachments of tumors, just as Langenbeck[13] had much earlier pointed out in his well-known *Beiträge z. Chir. Path. der Venen.* This is fortunate, since the vagus and phrenic nerves remain apparently uncompromised by a degree of pressure which, *a priori*, it would hardly seem as if they could resist. They permit, moreover, considerable displacement without interference of function."

Brodie[14] says: "If the function of the cord be interrupted above origin of the phrenic nerves, respiration is immediately suspended, and instantaneous death ensues."

Park[15] says: "Cases of unilateral division of the phrenic, or of injury to the course of the phrenic—as, for example, in fractures of the cervical vertebræ—indicate that paralysis of the diaphragm, or at least of one-half of it, puts the patient in immediate danger of life, and also compels the other accessory inspiratory muscles to do an enormous amount of compensatory work."

In a later work Park[16] says: "The section of the phrenic and pneumogastric nerves on one side only is accompanied by respiratory and circulatory irregularities; they are not necessarily fatal, but it is a most serious complication."

Tillmann[17] says: "In case of complete paralysis of the phrenic nerves on both sides, death results immediately from paralysis of respiration, as has been experimentally demonstrated upon animals. Irritation of the phrenic nerve causes continuous coughing and hiccoughing in consequence of contraction of the diaphragm."

Later on he says:[18] "After injuries of the subscapular nerve, reflex spasm of the phrenic nerve is sometimes observed with singultus, dyspnœa, and girdle pains."

Gowers[19] says: "If one nerve only is affected, the diaphragm does not descend on that side, but the movements of the other side lessen the resulting defect of movement, and it can then be detected only by close observation. The loss of the action of the diaphragm has little effect on the respiratory functions while the patient is at rest, but dyspnœa is said to be readily produced by exertion; the breathing then becomes quick and the voice feeble."

Rose[20] and Carless say: "Phrenic nerve division may cause instant death by paralysis of the diaphragm, although where but one nerve is divided the patient can survive. Irritation of the nerve gives rise to spasmodic cough or hiccough."

Medical men have not felt nearly so alarmed at paralysis of the diaphragm as have the surgeons, possibly because the lesion in medical cases comes on slowly, and in this manner patients may adapt themselves to new conditions with less danger. Thus Bernhardt[21] says: "The prognosis in paralysis of the phrenic nerves and diaphragm is doubtful, to say the least; but, as Duchenne[22] has shown, the respiration does not suffer much so long as the patient is quiet because of the action of the accessory muscles, but as one may readily understand, bronchitis and other simple diseases of the respiratory organs become serious in such cases."

Many other statements equally diverse in conclusion may be found in the literature.

TABLE II.

Case.	Surgeon.	Year.	Nature of injury.	Nerves.	Result.
1	Schürmayer,	1847	Stab wound.	Right.	Death in few minutes.
2	Beck,	1848	Bullet wound.	Left.	Death on fifth day.
3	Bardeleben,	1882	Unknown.	.....	Death in two hours.
4	Erichsen,	1884	Division while ligating subclavian artery.	.....	Death in few days.
5	Mackenzie,	1891	Rupture from fall.	Right.	Death immediate.
6	Schroeder,	1900	Division during operation.	Left.	Recovery.

I have been able to find only five cases of actual injury to the phrenic nerve (see Table II.), though there are several others which have been commonly reported as such which, when traced to the original documents, have been found to be erroneous. In some of these the injury to the phrenic nerve is only an assumption on the part of some later writer, who, noticing that the symptoms commonly described as caused by such an injury were present, has inferred that the phrenic nerve must certainly have been injured, while in still others the surgeon has reported an injury to the pneumogastric, but has been cited by others as having injured the phrenic.

The first of the cases of actual injury to the phrenic nerve is that of Schürmayer.[23]

Patient was a male, aged twenty-six years, previously well. He was wounded October 16, 1847, by a knife, which penetrated the right side of the neck. The wound began one and one-half inches above the acromial end of the clavicle, passed downward, forward, and inward, ending at the sternal end of the clavicle. In its course the knife-blade

injured the trapezius, splenius capitis, levator scapula, scaleni, and sternocleidomastoid muscles, the fifth cervical, phrenic and branches of the sympathetic plexuses, and the internal jugular vein. Immediately following the injury the patient complained of lameness of the right arm, which hung loosely at his side. Hemorrhage was quite severe. He left the room, going into the kitchen, and after a few complaining words, collapsed and died in the course of a few minutes.

The post-mortem, which was very complete, showed anæmia of vessels in skull and brain, but unusual fulness of vessels in lungs. There were numerous adhesions of the pleura.

Schürmayer says that death was not due to hemorrhage, because of the negative post-mortem findings, but that it was due to division of the phrenic nerve complicated by these numerous adhesions of the lung to the pleura. He believes it probable that cases of unilateral division may recover, and refers to Larrey, but declares that, in the sense of the law, death in this case was due to injury of the phrenic nerve.

The second case, reported by Beck,[24] was that of a young man who was shot in the neck during a duel. Upon examination it was found that the bullet had entered two fingers' breadths above the left clavicle at the posterior border of the sternocleidomastoid, and came out on the opposite side at the junction of the upper with the middle third of the neck, anterior to the sternocleidomastoid muscle. The respiration was slow and difficult, pulse weak, pupil of the left eye contracted. On the fifth day he breathed nine to seventeen times per minute. Laryngotomy was performed, during which operation the patient died. Beck performed the post-mortem and found the following: The bullet had contused the left phrenic nerve so that there was an extravasation of blood in the neurilemma, had passed back of the left carotid between the vagus and sympathetic nerves, lacerating the cervical ganglia of the latter, and had then passed through the œsophagus and lacerated the right arytenoid cartilage and the right descendens noni.

In conclusion, Beck states that as the injury to the phrenic nerve resulted in a partial loss of the function of the diaphragm, it certainly complicated a condition.

In some way this case has been attributed to Stromeyer,[25] who reported it about 1870, at which time he also reported another case of contusion of the phrenic and brachial plexus with rapid recovery.

In 1882 Bardeleben[26] reported a case of division of the pneumogastric and phrenic nerve with no immediate symptoms, but soon the patient's respiration became slow and difficult, and he died in two hours.

In 1884 Erichsen[8] says: "In a case with which I am acquainted, where the phrenic nerve was divided during a ligation of the subclavian artery, death resulted in a few days from congestion of the lungs."

In 1891 Mackenzie[27] reports a case of rupture of the right phrenic nerve with instant death.

In reviewing these five cases it will be seen that in only one, that is the last one, was there an injury to the phrenic nerve alone, while in

all the others there were sufficient injuries to other important structures to account for the unfavorable termination.

In considering the other cases, *i. e.*, those commonly but erroneously considered as phrenic nerve injuries, there are two which deserve special mention, as they have been so widely and frequently quoted, *i. e.*, Bransby Cooper's[28] and Hutin's.[29]

It has been commonly reported that Bransby Cooper, in 1841, while ligating a subclavian artery for aneurism, included the phrenic nerve in the ligature. In reviewing this case carefully, it was found that Cooper states that the patient developed a cough, which persisted more or less violently until his death on the fifteenth day. But the post-mortem, which was incomplete and was done fifteen hours later, showed the phrenic nerve "*in situ* and glued down to the scalenus muscle." Aspiration showed pus in the pleural cavity on the same side.

Hutin's case was operated on in October, 1841; for wound in the axilla. He got up on the sixth day, after which he began to cough. At this time the wound was fetid. On the ninth day he had a slight hemorrhage, and on the tenth day, for fear of a great hemorrhage, Hutin ligated the subclavian artery, but the patient died ten hours later.

Post-mortem showed the artery intact, and Hutin specifically states that no nerves or other important structures were found in the ligature.

Of five cases to which Park[15] refers as injuries of the phrenic nerve, four, three of Demme's[30] and one of Riedel's,[31] were found to be injuries of the pneumogastric, while in the fifth (Hofmokl's)[32] the injury was in the cervical plexus below the phrenic nerve.

Referring to the case which came under my observation, one might infer that as the tumor was of slow growth, the gradual pressure upon the nerve had produced a slow paralysis of the diaphragm; but in answer to this, I can say, as Langenbeck[13] and Diebel[12] have previously shown, that these nerves withstand pressure without much disturbance; secondly, upon pinching the nerve four different times, the epigastric region on the same side showed a sharp sudden rise each time, showing that there had been no paralysis; lastly, the physical examination before the operation showed the diaphragm to be in its normal position and the lungs absolutely normal.

Anatomical Researches. The subject of the innervation of the diaphragm will be considered under two heads.

1. Innervation from the phrenic nerve. 2. Innervation from any other source.

Under the second heading will be considered: 1. Innervation from the intercostal. 2. Innervation from the sympathetic. 3. Innervation from the pneumogastric.

Innervation from the phrenic nerve. The phrenic nerve or the internal respiratory nerve of Bell is the most conspicuous nerve supplying the diaphragm. It has, by most anatomists, both descriptive and comparative, been considered the sole nerve supply to this muscle. This nerve will be considered under three heads.

1. Its origin: (*a*) spinal or deep; (*b*) cervical or superficial. 2. Course and relations. 3. Distribution.

Spinal Origin. Marinesco,[33] in 1898, described a nerve centre in the spinal cord of dogs and rabbits, from which proceeds the nerve fibres of the phrenic nerve.

He describes it as a "group of cells which occupy an intermediate position between the anterior internal and anterior external groups of the anterior horn, extending from the level of the lower border of the fourth cervical vertebra to the lower border of the sixth cervical in dogs, and a little lower down in rabbits. In rabbits these cells are not distinguishable from other cells. In the dog they are smaller and their processes less marked."

Sano,[34] in the same year (1898), described a motor nucleus for the phrenic nerve in man, located in the centre of the anterior horn, extending from the middle of the third to the sixth segment. Sensory neurons originate, he says, in ganglia in the posterior roots of the third, fourth, fifth, and sixth cervical nerves.

Superficial Origin. Most of our standard English anatomical authorities give the origin as principally from the fourth cervical, with accessory branches from the third or fifth, or both, but with no figures as to relative frequency of different origins or the proportion in which different roots are found.

Luschka,[35] in 1853, published the results of thirty-two dissections of the phrenic roots, showing the following results:

Number originating from the 4th alone	12	37.5	per cent.
" " " 4th and 5th	7	21.87	"
" " " 3d, 4th, and 5th	7	21.87	"
" " " 3d and 4th	6	18.75	"

Brook,[36] of Dublin, in 1887, reviewed Luschka's work and reported sixteen dissections as follows:

Number originating from the 4th alone	4	25	per cent.
" " " 4th and 5th	9	56.25	"
" " " 3d, 4th, and 5th	3	19	"
" " " 3d and 4th	0		

In the past four months, from observations made in the dissecting-room of the Northwestern University Medical School I have obtained the following results:

Number of cases observed	52
" " on the right side	25
" " on the left side	27

These nerves had the following origin:

Number of cases from the 4th alone	9	17.3	per cent.
" " " 4th and 5th	21	40 38	"
" " " 3d, 4th and 5th	13	25	"
" " " 3d and 4th	9	17.3	"

These various results when tabulated show as follows:

No. of cases.		*4th only.*		*4th and 5th.*		*3d, 4th, and 5th.*		*3d and 4th.*	
Luschka,	32	12	37.5 per ct.	7	21.87 per ct.	7	21.87 per ct.	6	18.75 per ct.
Brook,	16	4	25 "	9	56.25 "	3	19 "	0	0 "
Green,	52	9	17.3 "	21	40.38 "	13	25 "	9	17.3 "
Total,	100	25	25 per ct.	37	37 per ct.	23	23 per ct.	15	15 per ct.

Number of roots. When there is a single root it is from the fourth cervical. This occurred in 25 cases in 100. When there are two

FIG. 9.

1, 2. Phrenic nerve. 3. Subclavian vein. 4. Internal jugular vein. 5. Common carotid artery. 6. Roots of brachial plexus.

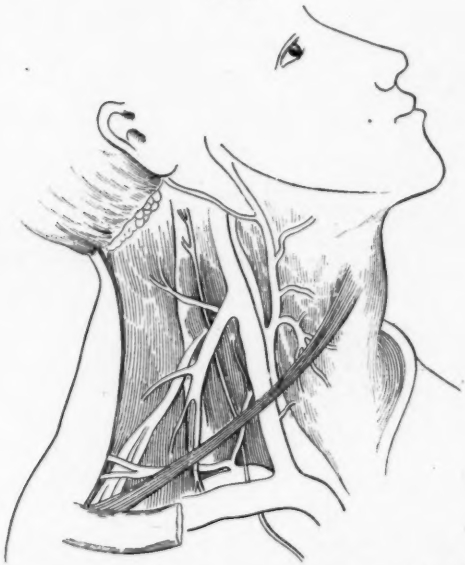
roots they are from either the third and fourth or the fourth and fifth. In 52 cases the nerve had two roots.

In 23 cases the nerve had three roots of origin, viz., from the third, fourth and fifth.

Course. The nerve seems surprisingly constant in its course. It is generally as described in the standard text-books, with few variations. One or two relations, however, are worthy of notice.

To the subclavian vein. I have noted this relation in 51 cases. In 26 cases on the left side, the nerve passed behind the vein in all. In 25 cases on the right side, the nerve passed behind the vein in 22, in front in 2, and in the remaining case passed through the vein. (See Figs. 9 and 10.) Of this peculiar anomaly I have been able to find but one similar case reported in the literature. In Virchow's *Archiv* for 1872, Von Wenzel Gruber,[37] of St. Petersburg, reported a case of the left subclavian vein dividing and reuniting so as to form "an island four millimetres long and two millimetres wide, through which passed

FIG. 10.



Course of phrenic (right) in neck, showing relations to adjacent structures.

the left phrenic nerve." Henocque and Eloy, in their article on the phrenic nerve, in the *Dictionnaire Encyclopedique des Med. Scien.*, say that Louget has observed a similar case, but I was not able to find any description of it in his work on the *Nervous System.*

Fawcett,[38] Barton,[39] Walsham,[40] Hunter,[41] and Turner[42] have reported one case each of the phrenic passing in front of the subclavian vein. Of these five cases, three were on the left side and two on the right. It is probably comparatively common.

I found this anomaly present twice in 51 cases, or nearly 4 per cent. It seems worthy of note on account of its relation to the clavicle. When

lying in this anomalous position it is immediately behind the posterior surface of the clavicle, and might be lacerated or divided in fracture of the clavicle or included in the callus.

Relation to the Internal Mammary Artery. At the origin of the artery: This relation was observed 22 times on the left side and 20 times on the right, or 42 times in all. In 22 cases (9 right; 13 left) it was external to the origin of the artery. In 13 cases (6 right; 7 left) it was internal. In 7 cases (5 right; 2 left) the nerve passed directly over the origin of the artery.

Relation of the nerve to the course of the artery. This relation was observed in 32 cases, of which 15 were on the right side and 17 on the left side. On the right side there were 14 cases in which the nerve was internal to the artery and one in which it was external. In 17 cases on the left side, the nerve was internal to the artery in 13 cases and external in 4 cases. In a total of 32 cases observed, then, the nerve lay internal in 27 and external in 5 cases.

Distribution. The main distribution of the nerve is to the diaphragm. Small twigs, varying in number, are sent to the superior vena cava, the pleura, and the pericardium. Luschka has described branches to the right auricle. This observation I have not been able to confirm. The branches in the thorax are small and easily destroyed, hence were not demonstrable in many cases on account of the parts having been disturbed before observations could be made. The branch to the superior vena cava from the right phrenic is very constant, and can, I think, be demonstrated in all cases where the integrity of the parts has been preserved. Branches to the pleura and pericardium vary in number from 1 to 4 to each structure.

The course of the main trunk of the nerve is as generally described, the left phrenic being longer and having a course far to the left over the pericardium. It breaks up into its terminal branches about 3 or 4 cm. above the thoracic surface of the diaphragm. It may remain intact until it pierces the diaphragm and divide in its substance or on its lower surface. In 29 cases in which the point of division was noted, the nerve divided above the diaphragm in 18, and in the substance of the muscle in 11 cases.

The statement made in some of our standard English works on anatomy, that the right phrenic passes through the opening for the inferior vena cava, is not borne out by the observations made. In the great majority of cases the nerve passed through a separate opening a few centimetres in front and to the right of the opening for the vein.

The diaphragmatic branches are from 2 to 6 in number, and are distributed to the lateral and anterior portions of the muscle, dividing and subdividing into fine nerve filaments, and soon losing themselves in the substance of the muscle. The exact areas supplied are as yet undeter-

mined. The single case of a dog observed by Dr. Schroeder, in which section was made of the upper roots of origin of the phrenic, the lower being unimpaired, is the only case of the kind I know of. In this dog post-mortem showed degeneration of the anterior and middle portions of the muscle, with the lateral portion normal. I hope at some future time to make some further observations on the exact distribution of these terminal branches.

In 15 cases the right phrenic perforated, in 12 cases branches were observed below the diaphragm, and in 9 cases it was possible to trace branches to the solar plexus. Two very careful dissections on unusually favorable cases showed branches of distribution as described, viz., to the semilunar ganglion, to the suprarenal and hepatic plexuses. These branches of communication in every case observed came from the innermost terminal branch of the right nerve, which pierced the diaphragm close to the inferior vena cava and ran downward and inward, retroperitoneally, to the semilunar ganglion. Whether the branches of distribution in the abdominal cavity are mainly from the phrenic or the sympathetic, or whether the branch connecting the right phrenic with the solar plexus is an afferent or an efferent branch, is as yet undetermined. In no case were abdominal branches observed on the left side.

The question of decussation of nerve fibres is undetermined, and upon this we have conflicting evidence. Kohnstamm,[43] in his *Studien über den Phrenicuskern*, says: "Innervation of diaphragmatic halves is distinctly bilateral, inasmuch as each phrenic supplies only the same side of the diaphragm and comes only from nucleus of same side, which in the rabbit lies from 6 to 10.4 segments of cord. A crossing of peripheral nerves in the anterior commissure does not occur in the phrenic. Ventral part of diaphragm is supplied by a cranial trunk and dorsal by a caudal trunk."

On the other hand, Henocque and Eloy[5] say: "The peripheral branches of the nerve (phrenic) form a plexus which completes the anastomosis between the two median halves of the diaphragm."

The question of nerve communication between the two median halves of the muscle is most important, but as yet undetermined.

Innervation from the Intercostal Nerves. Luschka,[35] in 1853, published a monograph on the phrenic nerve, in which he says: "The fleshy portion of the diaphragm receives motor filaments from the intercostals from the seventh to the twelfth." Later on, in his *Anatomie des Menschen*," he says, in describing the last two intercostal nerves: "During their course along the last six ribs these nerves supply not only the intercostal muscles, but also, as I have previously described, the costal portion of the diaphragm. These penetrate into the costal digitations of the diaphragm in company with the terminal branches of

the arteries, and are usually so fine as to consist of primitive nerve-fibres."

Previous to Luschka, however, Bauer,[45] Meckel,[46] and Valentine had described branches to the diaphragm in man, coming from the lower intercostal nerves. Their exact origin and distribution had not, however, been determined. Their work, however, seems to have been generally overlooked, or, at least, not emphasized. Cavalié,[56] writing in 1896, says: "They are not mentioned in any of our standard authors, except a foot-note in Beauner and Bouchard's *Anatomie*."

Among English authorities also these branches seem to have been overlooked. Quain,[47] in his last edition, refers in a foot-note to Luschka's work in a general way. McClellan[48] simply says that such branches exist. Gerrish and Morris make no mention of them. Gray[49] does not refer to them previous to the thirteenth edition, into which is interpolated the statement that filaments from the intercostals have been traced to the diaphragm. In most comparative anatomies there is no reference to this nerve supply. Chauveau,[50] Mivart,[51] Weidersheim,[52] Wilder and Gage,[53] and Huxley[54] make no mention of it.

In 1888, Pansini,[55] of Naples, described a subdiaphragmatic plexus in rabbits formed by "the phrenic and also branches from the three intercostals." This plexus he subdivides into three: anterior, middle, and posterior plexuses. He also describes numerous ganglia in these plexuses, especially in the posterior one.

In 1896, Cavalié,[56] of Toulouse, published the results of the dissection of six adult human diaphragms, or 12 lateral halves. By dividing the diaphragm in the median line and sawing through the spinal vertebra longitudinally he removed each half of the diaphragm intact, with its costal and vertebral attachments. After macerating the part in a weak solution of nitric acid, he demonstrated diaphragmatic branches from the intercostal nerves, with the following origins:

1. There are no diaphragmatic branches from the sixth intercostal nerve.

2. Branches were observed coming from the eleventh, eighth, and seventh most abundantly, then from the tenth, twelfth, and ninth in the order named, the last being relatively poor in branches.

Course. Diaphragmatic branches arise from the intercostals just as the latter pass over the insertion (costal) of the diaphragm. They either pierce the aponeurosis (of the internal intercostal muscle) or pass through its interstices, and are directed toward the muscular fibres of the diaphragm. They are sometimes alone and sometimes accompanied by the branches of the intercostal vessels. They number 5 or 6 from each intercostal nerve.

Distribution. These diaphragmatic branches from the intercostals supply a narrow margin along the costal border of the diaphragm,

varying from 1 to 3 cm. in width. Cavalié was not able to recognize any anastomosis of these fibres with the terminal branches of the phrenic, nor did he observe any of the ganglia described by Pansini.

In later papers, published in 1898, Cavalié and Billard[57] report the results of two series of experiments. By the first[57] they demonstrated the existence of motor fibres in the diaphragmatic branches of the intercostals. In the second series[59] they proved, by physiological experimentation, that the intercostal nerves cannot assume the rôle of the phrenic after division of the phrenic itself.

Stimulation of the peripheral ends of the divided intercostals in the first series of experiments showed visible contractions of an area of about 3 cm. wide in dogs, and of a length about equal extending in the direction of the muscular fibres. Tracings of these contractions were feeble and of slight amplitude. In the second series of experiments the investigators endeavored to ascertain if the intercostal nerves could assume the rôle of the phrenic in innervating the diaphragm after section of the latter nerve. They decided that it could not, although diaphragmatic contractions on stimulating the intercostals were greater in a dog whose phrenic had been cut a month previous than in one where the phrenic had been divided just previous to the experiment. Instead of indicating any vicarious action of the intercostals, however, Dr. Schroeder is of the opinion, from his extended work on dogs, that this is due to a simple hypertrophy of the muscle fibres of the costal margin of the diaphragm following paralysis of all the rest of the muscle, and not in any way an assumption of the function of one nerve by another. This certainly appears more reasonable.

Cavalié's conclusion from his physiological experimentation, then, is that the diaphragmatic branches of the intercostal nerves cannot, in mammals, assume the rôle of the phrenic after division of the latter.

Later, in 1898, Cavalié[58] published the results of an extended series of observations on mammals and birds, with four additional human dissections, confirming his previous observations, and bringing his series of dissections up to twenty lateral halves. His observations on mammals were made on dogs, rabbits, guinea-pigs, and rats, on birds, on the cock, pigeon, sparrowhawk, and duck. His observations on dogs are most interesting, and his description of the various branches of innervation of the diaphragm very minute and accurate, but we can only give his final conclusions:

1. The intercostal nerves send branches to the diaphragm in birds and mammals.

2. In birds and mammals these branches contain motor fibres. They probably do in man.

3. They play a certain rôle in respiration, secondary in importance after the appearance of the phrenic.

4. Birds have no phrenic nerve. The diaphragm is principally supplied by the thoracic nerves which correspond to the intercostals in mammals.

5. As one leaves birds and approaches mammals the rôle of the phrenic becomes more important, and the area supplied by it becomes greater; while the rôle of the intercostals becomes less important, and the area supplied by them becomes smaller.

6. In rabbits, guinea-pigs, and rats the last five intercostals send branches to the diaphragm. In dogs the last seven, especially the eighth, ninth, tenth, and eleventh.

7. In man the last six, especially the seventh, eighth, and eleventh.

8. In dogs the area supplied is the costal margin of the diaphragm for a width of about 3 cm. In a man for a varying width of 1 to 2 cm.

I have reviewed Cavalié's work at length, as he has done the largest amount of work along this line and, so far as I know, his investigations have never been reviewed in English.

Sympathetic Nerve Supply. The innermost branch from the right phrenic communicates with the semilunar ganglion of the solar plexus. I was able to demonstrate this communication but nine times, but found it in all cases where careful dissections were made on undisturbed structures. Whether these nerves are afferent or efferent is still undetermined. In no case did I observe any abdominal branches from the left phrenic.

SUMMARY. Our conclusions may be summed up as follows:

1. From the clinical and experimental data it would seem that the diaphragm is not an essential muscle of respiration.

2. That as the symptoms commonly described as caused by an irritation on the phrenic were uniformly absent not only in the operation, but in all of the experimental work as well, it is safe to infer that they may have been due to something other than a simple injury to the phrenic.

3. That while from an anatomical point of view the diaphragm undoubtedly is enervated by branches from the intercostal nerves, this nerve supply is secondary to the phrenic and is insufficient to carry on the action of the diaphragm after a division of the phrenic.

4. That a division of the phrenic nerve, producing a partial collapse of the lower lobe of the lung on the affected side and an atrophy of one-half of the diaphragm, might predispose to infection of the lung or be followed by a diaphragmatic hernia.

5. That a division of one phrenic nerve in man, resulting in paralysis of one-half of the diaphragm only, is not necessarily fatal.

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A NEW FACTOR IN THE ETIOLOGY OF MALARIAL FEVER, INDICATING NEW METHODS OF TREATMENT.

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The now well-known facts that malarial fever is a parasitic affection and that its paroxysms are produced by the sporulation of successive groups of parasites in the blood compel us to reconsider some of our former ideas as to the influence of meteorological factors in the etiology of the disease. Especially is this true with regard to the *heat* of the sun, which has been so long accorded paramount importance as an etiological factor. That solar heat generates fever "miasms" by evaporating swamps, etc., is no longer to be admitted. The dreadful mortality among Europeans—especially of European soldiers marching in the sun—in tropical regions, may have been *partly* due to malarial fever, but no doubt *largely* due to "thermic fever" or insolation. Which of these two components has been most fatal in the past must remain unknown. During the month of August, 1896, there were more than two thousand deaths from sunstroke in nine cities of the United States.[1] The mortality in the tropics must be still greater.

That a certain elevation of temperature is necessary to keep alive and maintain the inoculating activity of infected mosquitoes is true for all seasons and places; but, further than this, there seems to be no scientific evidence that solar heat is in any way a factor in the etiology of malarial disease. The malarial parasite cannot be affected by external temperature, for it is submerged in a fluid—really a blood bath—of 98.2° F. in a healthy human "host;" and in a malarial patient, while the temperature may reach 104°–106° F. during the few hours of the cold and hot stages of a febrile paroxysm, it is, during the much longer period of the intermission, usually *sub*normal, between 97° and 98° F., or thereabout.

The white man is most liable to this disease, the black least so; but the black skin of the negro absorbs heat like a black coat; thus there occurs the anomaly of the hottest individual being the fittest to survive in regions liable to fever from alleged solar heat.

The island of Tahiti is situated almost under the thermal equator and only 18 degrees from the geographical equator; yet the Tahitians are "free from marsh fevers."[2]

Dr. Manson,[3] in his excellent treatise, says the more we know of these diseases the less apparent becomes the rôle of temperature *per se* as a pathogenic factor and the more important the tropical fauna.

Dr. Celli,[4] in his recent (1900) work, observes that temperature and malarial fever do not run exactly parallel; and he reproduces a table

of Tacchini's covering twelve years, from 1871 to 1882 inclusive, regarding fever and weather factors, in which we see "that in the year 1879, when a true malarial pandemic raged, the mean temperature for the months of July and August was the *lowest* of these years."

If the solar heat theory be untenable, may we not explain the undeniable relation between hot climates and malarial fever by eliminating the term "heat" and substituting that of "light?" Suppose we say it is the light of the sun, not its heat, that determines the periodic sporulation of parasites in the blood? I propose this idea as a working hypothesis, and the evidence in support of it will be comprised in the following statements:

1. *The accumulated experience and observations of centuries, which have been held to prove the agency of solar heat in causing malarial fever, may be held to prove the agency of solar light.* The light and heat of the sun are inseparable. The "burning" sands of Africa must also be "dazzling" sands. So with hot seasons; the cloudless skies of a hot, dry summer diffuse an intense and brilliant light. We cannot conceive how external heat can warm the parasite in human blood; but that light can penetrate the skin and act upon the plasmodium to promote its development—as light is known to act on other amœbæ—is quite intelligible.

2. *Paroxysms of intermittent fever will not, as a rule, take place at night, in the dark.* Numerous authors support this statement. Wood[5] says: "It is worthy of observation that when the anticipating and advancing paroxysm reaches the period of darkness, it is either apt to be arrested in its course or to leap over the night backward into the evening or forward into the morning."

Flint[6] remarks: "Paroxysms may occur at any time of the day. They very rarely occur during the night."

"It is *entirely unknown*," says Fordyce,[7] "upon what this depends; indeed, the observation has been little attended to by any author who has not frequently seen the disease." The successive paroxysms getting an hour or two later may, however, be explained by the patient (languid from the paroxysm of the previous day) lingering in bed an hour or two later than usual, thus depriving the parasites of an hour or two of morning sunlight; as Flint[8] tells us, "a paroxysm is sometimes warded off by taking to the bed before the hour."

3. *The relative liability and relative immunity of different races of men to malarial fever depend upon the relative translucency or non-translucency of their skin, and probably of their blood.* All agree that dark-skinned peoples, in which Welch[9] includes "Negroes, Arabs, Indians, and Tamils," enjoy a relative immunity; and this has never been explained except by "specific idiosyncrasy" (a term without meaning) or by acclimation. Moreover, hundreds of negroes in the United States have the

disease, notwithstanding their alleged idiosyncrasy. Will the relative transmissibility of the skin to light clear up this difficulty?

In recently examining over a hundred negroes of different colors in Washington, to determine the translucency of their skin (easily done by passing the rays from a pocket electric flash-light through the external ear), I was surprised to find that *light passed through the skin almost as readily as through the skin of a white man.* In only three of the whole lot did I find the skin *absolutely impenetrable* to light. These were very dark, though not as black as genuine Africans, and, while they had lived in malarial places and had been bitten by mosquitoes, they had never in their lives (all were middle-aged) had ague. The affirmative evidence of these few cases, however, becomes of little real value, since there were other individuals with *translucent* skins who had *also* never had the disease.

It appears, therefore, that some negroes are not more immune than white people, because their skin, though of darker hue to an outside observer, is really not much more impenetrable to light than the skin of a white man. If my theory be correct, future experiment in tropical countries, etc., may be expected to demonstrate that individuals enjoying immunity will be found to have skins that will not allow the transmission of light.

White men who become black from malarial melanosis (cases are reported by Falls[10] and Gordon[11]), and white races becoming black after living for generations in malarial countries, may be considered instances of conservative structural modification to suit new and abnormal conditions, thus conforming to the general principle of adaptation to environment common to living organisms.

Finally, no white skin is impenetrable to light, and immunity in a white person is, so far, unknown.

Whether the *blood* of the negro is too dark, in the tropics, to admit light may be worth investigation. Cartwright[12] gives numerous authorities who affirm that the blood of negroes *is* blacker than that of whites.

4. *In places where malarial fever prevails the disease is increased by bright, sunny weather and lessened by clouded skies.* Observations on this point are plentiful, but they have been ascribed to "salutary rains" instead of to a clouded sky. Jackson[19] speaks of fever-stricken troops being benefited by three days of rain, and of a camp "over which *fog hung* until late in the day being better off than another placed in a dry and elevated situation." Of Trinidad, Ferguson[13] says: "It always rains nine months in the year; if it only rained eight, or if at any time there was a cessation of the preserving rains, the worst kind of fevers were sure to appear." In Tacchini's table, quoted by Celli, the year 1879, in which the percentage of fever was *greater* than in any other of the twelve years tabulated, is shown also

to have had the *least cloudiness* of all those years. It appears, therefore, that when the light of the sun is veiled by rain-clouds and fog, the parasites get less light and their sporulation is retarded. Conversely, the brighter the light the greater the liability to fever; hence the great liability of "sailors and fishermen" (Osler[14]) and of "berry-pickers" (Welch[15]).

5. *It has long been a popular tradition that to prevent the occurrence of ague, or to forestall its recurrence when it has once occurred, it is advisable to keep in the shade and avoid sunlight.* Persistent traditions have a certain amount of evidential value. Manson tells us the peasants of Italy and natives of German East Africa believed *for centuries* that fevers were caused by the bites of mosquitoes. So, now, the popular idea that sunshine will bring on a "chill," and shade prevent it, may be really true.

Osler, Thayer, and others affirm that intermittent fever is spontaneously curable without medicine. But in these cases of spontaneous recovery the patients were, presumably at least, shaded by their hospital surroundings from the light of the sun. That rest, food, and improved nutrition on the part of a human "host" should kill parasites in the blood seems unreasonable; and that being housed in bed prevents sporulation of the parasite, because of protection from the sun's heat, would force us to the admission that while so housed a man's blood is *too cold* to allow sporulation. Protection from *light* seems to be the only constant factor by which the spontaneous recoveries become explicable.

6. *The malarial parasite is a naked amœba. Red light promotes the vital activities of amœbæ, while violet or purple light restricts them. The color of the light diffused through the blood is necessarily red.* Experiments of Harrington and Leaming[16] on the common amœba proteus show that its protoplasm may be set in motion, or brought to rest, by varying the *color* of the light to which it is exposed. Their concluding summary is as follows:

1. Amœba streams in the presence of red light.

2. Streaming is retarded, stopped, or reversed by rays from the violet end of the spectrum.

3. Further, the effectiveness of the following kinds of light as inhibitors of protoplasmic flow diminishes in the order named—white, violet, red.

4. Enucleated amœbæ stream in red light and cease to stream in violet or white light.

If the plasmodium malariæ be a light-loving organism, red light would seem to be its natural requirement while in the blood, and we might expect to find its activities inhibited, like amœba proteus, by light from the violet end of the spectrum. This, in fact, we *do* find. Prussian blue (an old remedy for ague), and recently methylene blue, seem so far to inhibit protoplasmic movement in the malarial parasite as to pre-

vent its sporulation and thus cut short the ague paroxysms. Cases have been reported[17] by Mya, Thayer, Boinet, Thintignan, Huddleston, of New York, and Mühll, of Basle, and others. If methylene blue stains the parasite or its nucleus, this blue, commingling with the red of the blood, would produce the (to the parasite) disastrous violet or purple.

Possibly the (hitherto enigmatical) curative action of sulphate of quinine may be due to its remarkable *fluorescence*. In solution it intensifies the violet, and even renders the ultra-violet rays of the spectrum perceptible to human vision.

Of the few vegetable products having this quality of fluorescence, another one is esculin, the bitter principle of the horse-chestnut tree bark (*Æsculus hippocastanum*). But this also has been successfully used as an antiperiodic for intermittent fever.[18] Do these fluorescent substances act by intensifying violet rays in the blood?

If the etiology now given be correct, treatment is self-evident, viz., keep malarial patients in the dark, or in rooms with purple or indigo windows, and clothe them with garments impenetrable to light; in the tropics, with white clothing lined with purple or black. Give drugs that darken the blood or render it violet, or lessen its translucency.

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ANGINA PECTORIS.

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Angina pectoris, in its typical form, is in my experience a very rare disease. Pseudo-angina, or what resembles it at times, cardiac asthma, is not infrequent. Although angina pectoris is described among the neuroses of the heart by authors, this view in my judgment

is frequently erroneous. In the great majority of instances where angina is present there exist also organic changes of the coronary circulation, of the cardiac muscular fibres, or a lesion of the aortic orifices.

I should be loath to admit, except in a very exceptional way, the existence of true angina purely of neurotic origin. On the other hand, pseudo-angina is very frequently of this provenance, accompanied by symptoms of flatulent dyspepsia. It must be understood, however, that there are cases on the border-line in which, during life, we have great difficulty in pronouncing as to what symptoms are of nervous origin and what are clearly due to organic changes of the heart or arteries.

In many such instances, unfortunately, even if death occurs, we are not always able to obtain verification of clinical facts by the results of an autopsy. Hence, certain theories are brought to the front which have no basis in actual, reliable observations from the dead-house.

One statement is certainly true of angina, viz.: that it has usually connected with it an element of spasm or sudden intracardiac pressure which separates it notably from conditions which, in many ways, are similar, and yet from this stand-point differ manifestly in a greater or less degree. Of the truth of this statement we shall be convinced later.

In general, it may be stated that pain, properly speaking, does not characterize organic disease of the heart as we commonly meet it. Of course, there may be more or less præcordial anxiety or oppression, or there may be pain in the vicinity which is of stomachal or hepatic origin; but acute cardiac pain is very rare. For this reason it has become almost an axiom for clinicians to say when acute cardiac pain is complained of, it is more than likely no organic disease of the heart is present. Perhaps this affirmation is too positive or dogmatic, since I am confident functional distress may occur which is dependent upon obvious tissue changes.

In true angina the pain is very characteristic, and as I have said, ordinarily means organic changes of the heart structure. First of all, the pain of angina is marked by its great intensity. In no other disease, perhaps, is this so true. When the anginoid attack occurs, if the patient is walking or exercising in any way, he stops immediately and holds himself as quiet as possible, only taking hold almost involuntarily at times, so great is his distress, of the nearest object which will give him support. Otherwise, he may merely stand rigidly, having come to a short stop, with his arms lying unconsciously by his side, or else one hand is pressed firmly in the præcordial region, as if to ameliorate in a measure the subjective agony by so doing. During the attack the pectoral and other muscles of respiration scarcely move, and, indeed, breathing comes almost to a stand-still for a while. In this we perceive at once the great dissimilarity with an attack of cardiac

asthma, in which the efforts to breathe are so forcible and striking. And yet there are numerous instances in which the cardiac asthmatic features are most notable and where the "*angina sine dolore*" of Gairdner is also present,[1] as Osler states, after a masterly summary of the differential diagnosis of these two states, "when we recall to mind the features of the attack in cardiac asthma and in certain anginal seizures, the similarity of the condition, as Huchard remarks, to an acute emphysema, the views of Von Basch[2] appear to possess at least a reasonable probability" (p. 85).

The locality of the pain in angina is not always the same. Frequently it is located over the præcordial region. At times, however, it may be situated in the upper portion of the chest, or again, but in relatively few instances, it may stretch like a heavy bar across the xiphoid cartilage and the adjacent structures. Under these circumstances the pain may radiate directly through the chest and be felt even in the back. The pain has been likened to a heavy weight or crushing pressure, as though the thoracic parietes must almost meet. The pain is also said to resemble that of some terrible griping, as though the heart were firmly held in the clutch of some terrible monster. All these sensations, and numerous others, have been described and dwelt upon by writers, who doubtless have used their imagination at times to supply the descriptive powers of the patient. Suffice it to add that the pain is of a frightful sort and quite unlike any other we are familiar with. Often the radiation of the pain is toward the left arm, and in that case is usually carried through the forearm also and to the fingers. The ring and little fingers are said to be usually affected. Very rarely the pain extends to the right arm. When it does it radiates likewise in one or other direction mentioned.

According to Broadbent, the pain of angina often originates in the left wrist, and from there travels upward through the left arm and toward the chest—or again, as Osler states, although originating in the chest,[3] "was felt very severely about both wrists." This must be an extremely rare expression of the pain. I have never met it, nor do I find it mentioned by others. It is clear that the relations of brachial symptoms[4] (neuralgia) to angina pectoris are various. Sometimes, though infrequently, there are no pains in the arms, even in quite severe attacks of angina. Again, the brachial symptoms are very prominent, begin the attack of angina, and last longer than it does. The fact is, however, that the description of pain and its radiation, especially where it has the remarkable intensity of that belonging peculiarly to

[1] Osler. Angina Pectoris and Allied States, p. 82.
[2] These are: "Cardiac dyspnœa follows swelling and diminished elasticity in the lungs."
[3] "Angina Pectoris and Allied States," p. 42.
[4] Boston Med. and Surg. Journ., March 14, 1901, p. 256-57.

angina, must be somewhat inaccurate at times. In any event, it could only be obtained after the attack has passed, and I am inclined to believe that only exceptionally the patient could give any graphic and truthful description of it. What is literally true is that the patient has the impression vividly marked of impending dissolution, and it is this sense, together with the character, site, and evident intensity of the pain, which are almost pathognomonic of angina.

I know of few things more remarkable in descriptive medicine than the account given in the life of Dwight L. Moody by his son, of the anginoid attack near the close of the life of the great evangelist. In this case there was no terror or mental distress, as I believe, because his faith and works fixed him, as it were, on a rock. But in very many instances there is unquestionably great terror and mental distress. This is pictured often in the countenance which has that gray, ghastly, drawn look which once seen leaves an indelible impression upon the observers who may be near. The different radiations of the pain in angina are, no doubt, reasonably explained by the position of the cardiac plexus. The site of this plexus near the heart would serve to strengthen this view. In addition, we have its divisions and communications which appear to justify this interpretation.

According to one eminent observer, the pain originates probably in the central nervous system. This writer also explains its radiation and extension by affirming that it proceeds from the spinal cord. One thing is pretty certain, if we may judge by the few thorough observations we have recorded of these cases, and it is that there is no pressure outside of the heart on the plexus from any form of aneurism or other kind of tumor. While the heart may or may not be enlarged according to circumstances, this enlargement, even though present, does not explain rationally the anginoid symptoms.

Cardiac hypertrophy and cardiac dilatation are very frequently met with, and yet in the vast majority of these cases there are no anginoid symptoms properly speaking. Leaving aside these instances, there are a few where the heart is seemingly of normal size and volume, and we must, therefore, seek an explanation of anginoid pains in some other direction. Even in acute cardiac dilatation, no intense pain is felt, and yet we should have in just such cases pressure on or distention of nervous fibres under the endocardium. Moreover, this pressure or distention must be very much greater in patients thus affected than in those suffering with angina where no similar condition exists. During the attack the patient is usually very pale, and the pallor has a certain gray, ashy hue which is indicative of the serious condition which occasions it. Together with this pallor there is extreme weakness, and a faint feeling, which cannot be resisted, overwhelms the individual who is attacked. The pulse shows by its character, oftentimes upon what

this weakness in part depends. It is frequently small, feeble, irregular, as though the poorly acting heart could not send the blood to the extremities. Again, singular to say, it is almost unchanged, at least so far as we can appreciate by our tactile sensations. In the former instance, it is probable that there is present a spasmodic contraction of the peripheral arteries; in the latter, we must assume that no such spasm exists, or, indeed, that arterial changes are so advanced that no marked impression is made upon their contractibility even by the most intense pain and disturbance of the central organ of circulation.

Frequently an attack of angina terminates by a sudden explosion of gas from the stomach. Hence, it is often stated and familiarly accepted that flatulent dyspepsia is an immediate and efficient cause of a true attack of angina. In my experience this is scarcely true, and I am more inclined to the belief that it is especially in cases of *pseudo*-angina that we should expect to find symptoms of stomachal weakness or intolerance. While admitting this, we should also not completely ignore the fact that the stomachal conditions which occasion flatulence may at times appear to be of considerable importance, taken with other exciting factors, in bringing on an attack. There may be, as Broadbent points out, a certain sympathetic relationship between the terminal fibres of the vagi in the stomach and those in the heart. Many facts would serve to demonstrate this possibility. Certainly, even in cases of marked cardiac weakness, where there has never been a true anginoid attack, dyspepsia of an acute and very distressing type will frequently follow undue fatigue of any sort or any severe shock to the nervous system such as distressing or alarming news may readily excite.

A phenomenon which is somewhat curious is the fact of an intense desire to urinate during the period of an attack, even though the effort is vain, simply because the bladder is frequently entirely empty. This statement may not invariably be true. I have known many a time emotional excitement to prevent absolutely for a while the contractile power of the bladder being exerted, and where, as was proven later, the bladder contained a considerable quantity of urine. It requires a very slight degree of annoyance or mental disturbance in men past middle life to prevent frequently their power to void their urine. Of course, the contrary of this is true, especially among women of a neurotic type, and who are still relatively young. The quality of urine of low specific gravity passed by them at times, in a very brief period, is often very great. In the differential diagnosis of true angina with pseudo-angina, this point should be borne in mind. During the attack where it is severe, perspiration will flow almost constantly from the patient. His face and neck and hands may be covered with it. It is cold and clammy, and lends additional significance to the gravity of the other symptoms.

The time during which an attack lasts is very variable. Sometimes it is over after a few seconds, although during this short period the agony is fearful. Again, the attack is prolonged for several minutes. Some authors state that the attack may occasionally last throughout an entire night, and that during all this time the patient is unable to move at all on account of the intense pain, and, moreover, is perspiring profusely the entire period. I must confess that I have never seen any attack of this sort, and am inclined to consider them very infrequent.

It is highly probable, moreover, that in a case of true angina depending upon advanced degeneration of the coronary circulation, which would probably be present under such circumstances, the intense pain of anginoid character would terminate life more rapidly. Here, again, I should be disposed to hold the view that a neurasthenic or hysterical element was present, which gave strength and exactness to the true diagnosis, viz.: pseudo-angina.

It must be admitted, of course, that there are instances in which the attacks are certainly anginoid in character, although they do not reach their complete development. This fact may be explained by stating that the patient, having suffered from attacks previously, so soon as he fully appreciates that one is coming on simply stops still and avoids all possible exertion until the attack has completely passed away. In these examples there may be pain in the chest, but without radiations toward the arms or fingers. Of course, if the pain is diminished in violence, there is less dread attached to the seizures, and the patient does not expect to die at any moment. Broadbent and others speak of anginoid attacks *sine dolore*. In such instances the chief danger arises from a syncopal attack in which a patient may suddenly expire. It may be that these attacks had been originally painful, and it was only subsequently that they lost this characteristic feature entirely. I should expect, in such an instance, to find at the autopsy either marked fatty degeneration of the heart walls independent of coronary changes, or else advanced aortic regurgitation. Wherever the coronary circulation is suddenly obstructed with an embolus or thrombus, the breast pang seems to be almost an invariable accompaniment. In those cases where the attack has evidently been brought on by exposure or exertion, it does not usually last long, and when the accidental occasion of the attack has disappeared, the seizure itself is apt to dissipate itself rapidly. Wherever the attack comes on spontaneously, as it were, without any accidental efficient cause being evident, it is apt to last a longer time, and only to pass away little by little and slowly. Occasionally these attacks are the most alarming in reality, and herald a fatal termination in the not distant future.

Among the causes which act efficiently in bringing on an attack of angina are primarily exertion. We are apt to say over-exertion when

the attack has taken place. This over-exertion may be a brisk, rapid walk, or the patient may be walking leisurely and without effort when the seizure occurs. Usually, however, it is when a walk has been prolonged and there is already a feeling of fatigue that the angina is felt. It has been noticed that whenever exertion takes place soon after a meal an attack is more apt to occur. It may be because digestion, if slow and torpid, is thus interfered with, and gases which are generated and accumulate in the stomach press against the diaphragm and indirectly against the heart, and thus, by causing some displacement of this organ, may occasion notable interference with the circulation.

One reason, no doubt, why attacks occur at times during sleep, is because flatus is prone to accumulate in the stomach and intestines during sleep, and considering this together with the fact that in the horizontal position we have more pressure of the abdominal viscera upward, we realize readily conditions which are powerful in causing distress. The liability, under these circumstances, to an attack is increased notably by a feeble circulation. Moreover, as we know, the circulation is always less active in repose, and this state is what prevails during sleep. A loaded rectum is, also, a condition to be avoided, and anyone subject to constipation must see to it that the bowels are properly evacuated. The distended bowel may perhaps act in a reflex manner, as well as by direct pressure.

The influence of cold is sometimes very evident. This is particularly true when a patient is walking against a cold wind. Nothing more is required than this sometimes to precipitate an attack. On the other hand, mild, warm weather is conducive to well-being, and sufferers from angina will often escape attacks during long periods when the weather is free from rapid changes and remains relatively balmy. It is essential at night for a patient to see that the bed is comfortably warm and that no chilling of the surface ensues; otherwise, an attack will often follow. This precaution may be readily attended to with a hot-water bottle or heated bricks. Gentle friction of the surface of the body, perhaps, before the patient retires, is also a proper precaution to take. The wearing of long woollen stockings, and particularly those which are somewhat loosely knit and allow free transpiration, is especially desirable, so as to keep the extremities suitably warm. I know of no small detail so important as this in all affections in which the circulation is notably impaired, and, of course, it becomes doubly imperative in warding off painful attacks which are too frequently occasioned by local chilling of the feet.

In view of the fact that dyspepsia is such a frequent symptom of angina, and appears as an efficient cause, in the judgment of a few writers, quite as often as an effect, it is important to avoid all late or too abundant dinners. The food at this meal should be of the simplest

kind, and no overloading of the stomach should be permitted. In a similar way, no sauces, condiments, or insufficiently cooked food should be tolerated.

Whenever an attack has occurred, it behooves the patient to be more than usually circumspect in all his doings, not to bring on another one. This is especially true of any exertion which seemingly has been the direct cause of an outbreak. And yet this counsel is sometimes almost unnecessary, because the patient's own feebleness, which follows a primary attack, will compel him almost to walk very slowly and deliberately, even if he walks at all, for some hours or days subsequently. No doubt these anginoid attacks would not occur if the heart had sufficient reserve force to respond adequately to the call made upon it. Unfortunately, it has not, and it is therefore evident, in many instances, that the attack is directly occasioned when we reach final causes, by the manifest inability of the heart to respond to the call made upon it for increased vigor.

Angina pectoris is not necessarily connected with any special lesion. Practically, it is almost unknown to have either stenosis or incompetence of the mitral valve appear as a direct, efficient cause of it. It is true, however, when there have been several attacks of angina, it is not infrequent to observe mitral incompetence arise subsequently. In some of these instances it has been noted, where aortic incompetence already existed, that this affection was ameliorated as regards its symptoms, and that the anginoid attacks also became less severe. The explanation appears to be in the lessened blood pressure thus brought about, as shown in the arterial pulse and in the diminished accentuation of the aortic second sound. Musser[1] has insisted upon the importance of this finding, and has reported several examples in his own experience. Broadbent[2] has also specially emphasized similar instances.

Angina is frequently connected with fibrous myocarditis, and at the autopsy such organic change in the heart muscle is apt to be found. So usual is this condition that Gibson states it is almost a surprise not to find it. When fibrous myocarditis is noted, it is frequently accompanied with evidences of arterial degeneration. The coronary arteries are usually implicated. Especially is this true where aortitis is present. The lesion may be limited to their origin, which is sometimes narrowed and thickened. The arteries may also be affected in a considerable extent, and the organic changes may be considerably advanced. Occasionally they have merely lost elasticity; in more pronounced alteration they may have become markedly atheromatous, or, indeed, calcified.

According to Douglas Powell, fatty degeneration of the heart walls often exists. Sometimes, indeed, the heart is so much degenerated that

[1] Transactions of the Association of American Physicians, vol. x. p. 85.
[2] British Medical Journal, 1891, vol. i. p. 747. Quoted by Osler.

it is easily torn, and the finger sinks into it on slight pressure. Sometimes, to the naked eye, the fat exists only in patches, affecting merely the papillary muscles or different areas of the ventricles. Even in these instances, however, if we make use of the microscope, we are apt to find considerable degeneration of the walls, where there has been no real change of coloration. In a few instances the microscope shows almost complete disappearance of the muscular fibres.

As a concurrent condition with fatty degeneration, we discover more or less advanced changes of the coronary arteries, very similar to those already mentioned in connection with fibrous myocarditis. These evidences of fatty degeneration are particularly found, of course, where the fatal termination appears to be intimately dependent upon the previous anginoid attack. It should be remarked in this place that we often have both anterior conditions, viz.: that of fibrous myocarditis or of fatty degeneration, without having attacks of true angina. According to Gibson, the relations of angina with endocardial lesions is not so distinct. It is true, of course, that degeneration of cardiac walls may often cause it, and, therefore, it is frequently found at the autopsy. The earlier writers, like Morgagni, certainly attached angina directly to the existence of aortic disease, and in one of Heberden's cases, where the autopsy was made by John Hunter, this affection is duly recorded. On the other hand, we know that the most advanced changes with ossification may exist at the aortic orifice, and yet there may be present during life no morbid symptoms at all.[1] These instances must be somewhat exceptional. What is true is, where aortic lesions have been proven at autopsy, often pain has been noted prior to death. No doubt, this pain has been in part due, at least, to interference with the coronary circulation, caused by accompanying aortitis, with which there may be also a certain degree of dilatation, or, indeed, a sacculated aneurism. Arterial degeneration, especially arterio-sclerosis, is often adjoined to attacks of angina. Where the coronary arteries are degenerated, and where the angina is seemingly dependent upon this condition, we should not lose sight of the fact that the other arteries, being degenerated, are also doubtless contributory. The affection of the coronary arteries through sclerosis and consequent narrowness, prevents a sufficient blood supply reaching the heart, and hence interferes with its nutrition. A thrombus or embolus may obstruct the vessels, but it is doubtful whether one or the other of these conditions causes angina (Gibson). One thing is sure, viz.: we often find calcification of these arteries without previous anginal attacks. Adherent pericardium may be found, but does not occasion anginal attacks unless accompanied by a lesion at the aortic orifice.

[1] Semple, p. 104 et seq.

Angina has been observed following injuries to the chest walls. In these cases the aorta may have been affected. Broadbent states its presence occasionally in malarial fever. It may also be present in advanced diabetes where the arteries may become thickened, thus giving rise to increased tension, and followed by attacks of true angina. It is not infrequent to find anginoid attacks occasioned evidently by the presence of the gouty poison. In these cases the prognosis is only serious where the intracardiac changes are already advanced, as shown by the weakness and irregularity of the heart's pulsations. In some of these cases we find notably fibroid myocardial changes in patches or disseminated. Even in these instances, prior to death, there may have been few or no threatening cardiac symptoms. In a few rare cases neuritis of the cardiac plexus and also of the phrenic nerve has been noted (Lancereaux, Peter) where anginoid attacks have occurred. The view of Semple is, indeed, that angina considered as an idiopathic disease is connected with an affection of the pneumogastric or phrenic nerve. Of course, it is difficult always to pronounce what the precise structural changes are. Still, they are doubtless present, and later will be discovered. Meanwhile we are forced to rank a few such instances among the "neuroses." Frequently, doubtless angina is associated with minute structural changes which only subsequent close investigation will determine. Flint is evidently of the opinion that the connection between angina and organic lesions of the heart is rare. Thus he has only observed fifteen cases in 388 cases of the latter. Again, Flint says that in ten years he has noted only four cases of true angina, that is to say, where the disease was unconnected with cardiac or aortic lesions. It is clear that in all instances where there is present an organic lesion of the heart or arteries, whatever suffering, if any, the patient may experience should be directly explained by them. This leads to the conviction that only those cases in which no such lesion is discoverable should be ranked among the true cases of angina. As a rule, when sudden deaths occur in what has been called angina, pathological lesions sufficiently explanatory are found at the autopsy.

The condition of the heart during an attack of angina has been believed to be one of spasm. At least, this is the opinion of some writers. It is certainly true of Heberden, who first so accurately described these attacks in his commentaries. This does not seem to be altogether a tenable opinion, if one has regard to the fact that the heart has rarely been found thus contracted.

Usually the heart stops in diastole, and is found after death in a relaxed condition. Again, during life, while the pulse at the wrist is sometimes irregular and weak, it never disappears entirely, which it certainly would if the heart were in a state of forcible, spasmodic contraction.

It has been supposed that the heart during an attack presented a sort of hour-glass contraction not dissimilar to that of the uterus. This may be, and yet it would be difficult to prove. Broadbent confesses in this connection that he has a very imperfect notion of what the condition of the heart really is during an attack. The evident fact is that during a paroxysm of angina stress is put upon the heart to which it is quite unequal to respond, and thus it shows its considerable lack of power. Often the stress put upon the heart is due to the continuous high tension of the peripheral arteries. Occasionally, however, there is low tension in the peripheral circulation, and in these instances, if there occur a sudden general arterial spasm, the amount of work thrown upon the heart becomes rapidly much greater, and consequently the heart shows relatively greater distress than where the peripheral resistance is continuously high and exaggerated. In those cases where there is marked and continuous high tension in the peripheral arteries we might suppose that pain in angina was explained by greater pressure thrown upon the heart. This can scarcely be true when we consider how many such cases escape any such pain. Again, in acute dilatation of the ventricles, we have great pressure brought upon the heart walls, and yet no pain results. Neuralgic predisposition is occasionally given as an explanatory cause. This is scarcely true if we mean by that an acquired or evident neurotic tendency, since this disease occurs more frequently with men than with women. It would seem as though from the fact that when the attack occurs immediately, the patient stops doing anything he is occupied with, or exercising, if that be what he is about, that there is a certain pre-ordered protective arrangement internally to guard against these outward manifestations of man's life. (Broadbent.)

Pain, according to Bramwell,[1] is due to irritation of the nerve terminations in the walls of the heart itself. He admits, however, that the theory of irritation due to spasmodic contraction is plausible, and compares this opinion with what occurs in the calf muscles, spasmodically contracted, in ordinary cramp.

Attacks of false angina often resemble those of the real kind. Sometimes the description of the attack by friends will enable us to reach a correct diagnosis. If the patient becomes pale, anxious, and shows signs of great distress, it may not of necessity be true angina. If, on the contrary, there be no such changes evident, we can be very sure that it is only an attack of pseudo-angina. There is much unreliability in the patient's accounts, mainly because they are apt to read up about these attacks, and often give an exaggerated idea of their own sensations. Age will throw some light upon the diagnosis. Under forty

[1] Diseases of the Heart, p. 676.

years of age in the male, angina occurs very rarely unless there be a pronounced lesion at the aortic orifice or aortitis. In females it is rare at any age, although attacks of pseudo-angina are not infrequent with them, especially those of an undoubted neurotic temperament. Heberden, for example, in speaking of the cases observed by him, being in number over 100, states that three occurred in women, one in a boy, twelve years of age, and the others in men near or over fifty years.

Usually the first attacks of angina occur during physical exertion. Later on they may come on severely and more readily, and then we may make the diagnosis surely, even though the determining cause is slight. The physical examination will reveal, in case of true angina, the changes mentioned in the aorta or at aortic orifice. Where the attack at first comes on without exertion and at a fixed period after ingestion of food, it would seem to be of digestive provenance or pseudo-angina, and ordinarily due to a dyspeptic attack. Unless history, nature, and onset of attack all concur together with physical signs to establish diagnosis of true angina, we should lean strongly to diagnosis of pseudo-angina, and almost invariably, if we can discover any facts to support this diagnosis. If we leave out attacks of pseudo-angina which are evidently neurotic or of hysterical nature, we can usually find in some digestive disorder, particularly of the stomach, a sufficient explanation of them. There are eructation, marked flatulence, pain, occasional attacks of nausea or vomiting, which all point in this direction. A combination sometimes found is that of dilatation of the stomach, with high arterial tension. If the heart be affected in these cases and they are improperly treated, a fatal result may not infrequently follow. If, for example, digitalis or nitroglycerin be alone used, or the Schott treatment advised and carried out without any care of the stomach, and especially if the subject be old, such a denouement may not be a surprise.

The prognosis of true angina is often uncertain; and yet we have certain conditions which guide us to make it correct. We should estimate carefully the relative predominance of the two factors often producing it—on the one hand, degenerated heart walls, on the other, vascular changes. If there be high arterial tension present, and if at the same time the heart action is forcible and the aortic second sound marked, we may hope by proper treatment to modify these conditions for a while with the use especially of nitroglycerin and the nitrites. Again, if over-exertion and excitement bring on the attacks, or if flatulent dyspepsia be a decided and powerful influence in producing them, we should hope to avoid with care and treatment their natural outcome.

On no account should the patient walk hurriedly, especially in going up hill. He should also never take even a moderate walk until a certain time had elapsed after his last repast.

Attacks of angina which accompany aortic disease may last a considerable time without bringing on a fatal result if carefully watched and guarded. The worst cases are those which recur in the night or at times where no accidental cause is present and avoidable which occasions them. Again, if examination is relatively negative, if the heart is of normal size, without manifest lesion of any kind, and yet its action is feeble, its impulse scarcely felt, and the pulse usually, if not invariably, of low tension, these give great anxiety by reason of the vagueness and uncertainty as to the conditions which may be present and at any time become imminently threatening.

The apparent severity of two attacks may be similar, and yet the relative danger of them may be absolutely different. It is difficult, therefore, at times to make anything like a sure forecast. Of course, where there are pronounced cardiac and arterial changes, and where, in addition, heart failure has followed hypertrophy, the outlook is assuredly very grave indeed. If, at the same time, aortic regurgitation also be present the prognosis becomes even more serious. In a similar way, if chronic renal changes exist the future of the patient must appear dark and imminent. Gibson states that the prognosis of those affected with fatty degeneration is far less serious than the preceding. Provided always the external and avoidable causes of aggravation are prevented, such patients may often live many years. Of course, toxic angina is far less grave. As a rule, with the removal of the cause the case becomes curable. In neurotic cases, while we should expect frequent recurrences of the paroxysms, it is wholly improbable to have a fatal termination.

In this category may be placed frequently the so-called idiopathic cases. They are often extremely painful, but as no incurable lesions exist, they tend to improvement or recovery if properly managed. Of course, we should be careful in making even in these cases too favorable a prognosis, since there may be some underlying structural change of the heart walls or coronary arteries which, during life, could not be determined. There are unquestionably, according to Semple, certain cases of pure angina in which the autopsy reveals no organic changes.

The treatment of angina depends upon what is the apparent or obvious cause. In many instances, owing to the difficulty of tracing accurately to what the attack is primarily due, our treatment must be essentially empirical. First of all, we must consider the general health, and from this point of view our treatment should be hygienic. The means at our command are here what pertain to air and light, rest and exercise, food and drink. After these have all been inquired into and regulated, as far as may be, we naturally seek for the proper medicinal remedies to meet the indications of each special case. In general, also, the efficacy of our treatment will depend much to what degree we may

be able to relieve peripheral resistance to a heart frequently weakened. If, perchance, we find between the regular pulse beats, evidence of increased tension to our tactile sensations, we may often reduce this by appropriate remedies. Still, in order to recognize it, we must at times examine the heart and arteries at different periods, before and after exertion. Not infrequently the arteries are notably degenerated, hard, thickened, tortuous, and even calcareous. We can then do little to affect them directly. Yet, the capillary system, in which there may be notable resistance without excessive changes, and which has caused in a measure the arterial and cardiac changes, may be still favorably influenced by appropriate drugs, and account should be kept of this fact. In gouty conditions the peripheral circulation may show increased tension, although not visibly degenerated, and this condition, of course, may be favorably influenced by appropriate medication.

In these latter cases the ordinary treatment with a mercurial, followed by a saline, once or twice a week, will lower arterial pressure. Between times the use for a while continuously of iodide of potash and colchicum may be of signal benefit. The employment of bitter tonics, if the indication presents, and the proper regulation of the diet is of course useful According to Powell, hop, columba, and chiretta are better tonics in these cases than quinine and strychnine. In the "neuralgic bouts," to which they are prone, he praises quinine and phenacetin. Where angina occurs with marked aortic disease it is difficult sometimes to know to what extent we may be able to help the attacks by reducing tension of the pulse. Where the pulse remains feeble between the attacks, and the heart has a weak impulse, we should carefully endeavor to help with cardiac tonics, but frequently we can be of little real service in view of the pronounced degenerative changes present in the heart and arteries. Occasionally arsenic, combined with iodide of potassium and nux vomica, is useful where the arterial tension is not too pronounced. Preference may be given in many instances to the sodium salt of the iodide, both between and during the attacks. According to Schott, it is less prone to cause heart failure; but even this salt is "apt to destroy the molecules of albumin" if continued too long or in increasing doses. Milk is the best menstruum for either salt, as in this way stomachal intolerance is less likely to occur. In these and other cases we should try to preserve the use of the nitrites and nitroglycerin for the attacks. Formerly the diffusible stimulants, like brandy, ammonia, lavender, camphor, etc., were much used for these attacks. Now they are almost abandoned for nitroglycerin and nitrite of amyl. These latter are particularly useful in relieving pain, and to accomplish it they dilate peripheral arteries. Nitrite of amyl by the rapidity of its action is preferably employed. Nitroglycerin and the sweet spirits of nitre produce similar effects in different degrees.

All of these are free from dangerous effects, as a rule; not so of nitrite of sodium, which may produce alarming results (Gibson). Nitrite of amyl and nitroglycerin dilate arteries, increase frequency of pulse and respiration, and reduce irritability of the nervous system. Where increased acceleration of the pulse and respiration are already present the nitrites must be employed with great care, as they might possibly cause greater distress. While they are said to be heart stimulants, they mainly cause relaxation of the arteries and also of the cardiac muscular fibres (Broadbent).[1]

The nitrites have their drawbacks also in the fact that patients find so much relief from their use that they use them too frequently and injudiciously. A word of warning should be thrown out because life is sometimes shortened by their inconsiderate use. Glycosuria has been produced by them, it is stated. In many instances the nitrites are less useful than iodide of potash. Nitrite of amyl may be carried about with one so as to be used immediately. The nitrite of amyl in glass globules, of 3 to 5 minims, may be in a silk bag and broken upon a handkerchief and inhaled as required. The nitroglycerin tablets, one one-hundreth of a grain, may also be taken in doses of one or two when attack occurs. They do not act as rapidly as the nitrite of amyl, but their effect is more prolonged, and on that account may be more valuable in certain cases.

Some cases, however, are not relieved by nitroglycerin tablets and are relieved by nitrite of amyl. According to Broadbent, such cases have seemed to him to originate in the right ventricle.

It is the belief of Dr. B. Addy[2] that we have in erythrol tetranitrate a remedy superior even to "nitroglycerin," its effects being very rapid and more lasting. Tablets of one-half grain each were given by him twice or three times a day. They did not cause headache, and the remedy soon checked the attacks. It is true, the patient died after a fortnight of syncope, but during this period great relief from suffering was experienced.

Sometimes, where the heart is weak and the nitrites do not relieve, although they may relax the peripheral circulation, we must recur to the old stimulants. In addition, a turpentine stupe, or mustard leaf, or poultice may be applied over the chest and will occasionally afford a measure of comfort. Whenever these local applications fail, great relief is obtained from a hot-water bag at a temperature of 140° F. to 170° F., "moved with light touches over the whole chest."[3] If, despite all this, the attack is prolonged and unrelieved, we must give a hypodermic injection of morphine and atropine, using at first small

[1] This opinion about heart fibres I do not share save very exceptionally.
[2] British Medical Journal, May 6, 1899, p. 1089. [3] Lancet, September 8, 1900, p. 726.

doses, and later, if need be, becoming bolder, and using larger doses. It is well to make injections deep in the muscle, where the circulation is more active than in the cellular tissue under the skin. In some instances, we should recur to chloroform inhalations as being the only hope of relief to the patient. At times they are undoubtedly dangerous, and especially is this believed to be true if fatty heart is present. As a matter of fact, however, fatty heart cannot always be diagnosed with accuracy. The apex may be strong and the pulse regular and good, and yet fatty heart may exist, and sudden death follow. Again, moreover, it has been shown that chloroform may be given safely where fatty heart later is known to exist by the revelations of the autopsy. We must relieve intense pain, however, even if there be risk, and it can only be done at times by such agents (Balfour).

In cases where there is marked heart failure, ether or brandy should be employed hypodermically in doses of ʒss to ʒj. To each hypodermic injection one or two tablets of one one-hundredth of a grain of nitroglycerin may be added. The latter should be employed with caution, however, as occasionally considerable soreness and even ulceration of the skin may result.

Theodore Schott does not value very highly digitalis or strophanthus in cases where the heart requires stimulation, even in uncomplicated forms of angina pectoris caused by sclerosis of the coronary vessels.

Oxygen inhalations are often also useful, not only to satisfy the air hunger, due to obstruction of circulation in the lungs, but also to stimulate cardiac circulation and help nutrition of its muscle, and thus get rid of effete material which interferes with proper metabolism (Powell). In these cases the oxygen must be used with a funnel near the nose and mouth, so that it may be inhaled frequently and without effort. As corroborative of the extreme value of inhalations of oxygen in the treatment of some severe cases of angina pectoris, I would refer to one recently reported in the *British Medical Journal* for December 1, 1900, p. 1568.

Rest in bed for a time is often desirable after acute paroxysms have passed, but later it is useful, as far as possible, to get the patient back to his ordinary life with judicious restrictions. The same rules apply here, however, as in other heart affections. We must remember, also, that exertion which one day may seem all right, another day may cause distress and oppression. This is one of the objections to Oertel's system of treatment (Broadbent).

Physical therapy is undoubtedly useful in some instances, but it must be utilized with great care. This counsel pertains particularly at the present time to the treatment as instituted at Bad Nauheim, where the resistant movements in conjunction with carbonic baths are prac-

tised. In advanced arterio-sclerosis every increase of the blood pressure which is the result of this treatment might lead to fatal consequences (embolism, apoplexy, rupture of aneurism of heart, or aorta). "Advanced sclerosis is, therefore, a contraindication for this treatment" (Schott). The value of many medicaments in angina comes from producing low blood pressure. The balneological and gymnastic treatment exercises a tonic influence, and "by strengthening heart muscle, as well as by acting on cardiac nerves, distressing symptoms of angina are either removed or reduced" (Schott).

Guidance should be had in regard to the bad effects of winds, great heat or cold, or rapid changes. Also, an atmosphere heavily laden with moisture is injurious. Internal conditions of dyspepsia and constipation must be warded against. A great deal of tact and good judgment are required, and the patient's disposition should be thoroughly known. Rest, particularly after meals, should be insisted upon, as patients are particularly liable to attacks at these times.

In general, supervision and counsel must be employed about exercise. Where an attack has lately occurred, it is wisdom to refrain from exertion for a while, especially if the heart is weak and fluttering, and afford it time to re-establish itself.

THE PATHOLOGY OF THE HEALED FIBROUS ADHESIONS OF THE PERICARDIUM.

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In a total of 1048 autopsies upon the bodies of adults, the records of which are among those of the Pathological Laboratory of Rush Medical College (in affiliation with the University of Chicago) and of the Cook County Hospital, some inflammatory change was found in the pericardium in 128. These changes included everything in the nature of a pericarditis, from the frank serofibrinous and tuberculous processes to those with but a thin film of fibrin over a small area, or a fibrous band uniting the parietal and visceral layers at some point. Of these 128 cases 57 showed changes that were essentially chronic, the lesion consisting of fibrous tissue uniting the opposed surfaces for a greater or less extent. These figures agree closely with those of Leudet,[1] who found in 1003 autopsies adherent fibrous pericarditis 61 times. The remaining 71 were acute, and represented all the conditions usually seen in acute pericarditis.

Of the 57 cases with fibrous adhesions the cause could be attributed to rheumatism in 8; in 6 the lesion was frankly tuberculous, as shown

by the presence of caseous masses or miliary tubercles in the adherent pericardium. There remain 43 cases of complete or partial synechia in which the cause cannot be thus determined from the anatomical findings or the history—certainly an impressively large proportion; 24 of these 43 were cases of total obliteration; in 14 the adhesions covered but part of the heart, and in 5 more a single firm band united the apex of the heart to the opposite pericardium. The large number of such cases of dry, fibrous synechia of the pericardium, for the most part lying latent during life and found only at autopsy, directly calls attention to the exactly similar condition that is still oftener found in the adjacent serous cavities, the pleural. Synechia of the pleural sacs, either total or partial, is an extremely common occurrence, and was recorded in 63 per cent. of all the 1048 autopsies from which these cases were gleaned, which figure is undoubtedly too low. In all but 3 of the 43 cases of "idiopathic" pericardial synechia there were fibrous pleural adhesions also present—that is, in 93 per cent. Furthermore, in a considerable number of cases in which the pleural adhesions were limited it was expressly noted that the pleura was adherent over the external surface of the pericardium. In several cases the pleural adhesions, where abundant, were expressly stated by the examiner to be especially firm over the external surface of the pericardium.

From these observations it would seem probable that the cause of adhesive fibrous pericarditis is in many cases the same as that of adhesive fibrous pleuritis. In addition, it is quite probable that the pericardial changes are secondary to those of the pleura much oftener than the reverse is true, for being a closed sac the pericardium has but limited chances of infection. The possible sources of infection of the pericardium are: 1. Through the blood stream. 2. Through the lymph vessels. 3. Direct infection by trauma. 4. By extension, which must be from (*a*) heart or vessels within the pericardium; (*b*) mediastinum; (*c*) pleura; (*d*) peritoneum.

Infection from the bloodvessels means a bacteræmia, which is bound most generally to produce an acute process in the pericardium, which is usually fatal and does not often terminate in the formation of fibrous adhesions unless the infecting agent be rheumatism, tuberculosis, or syphilis. Rheumatism affecting the pericardium usually affects the endocardium also, and these cases and other cases with a history plainly indicating rheumatic origin for the trouble have been excluded in this series. Of our 43 "idiopathic" cases, but two, so far as the history sheets tell us, had ever suffered from rheumatism. Syphilitic infection of the pericardium is one of the rarest of its manifestations, and when present is usually secondary to a gumma of the myocardium, which leaves sufficient traces to explain the source of any adhesions that might result. Mracek,[2] who has investigated the literature on this subject,

says: "Syphilis has hardly ever caused an exudative serofibrinous pericarditis." A. Genersich[2] has recently reported such a case. Nor does syphilis often affect the pleura. Among the autopsies studied there were 35 cases of marked syphilis, but in none were the pleura or pericardium evidently affected by it. Tubercle bacilli reaching the pericardium, or any other place, by the bloodvessels produce almost necessarily distinct tubercles, so that should the resulting pericarditis heal—which cannot be common in a tuberculosis of vascular origin—the characteristic fibroid, caseous or calcareous nodules would usually give evidence of the source of the adhesions.

From the lymph vessels the most common infection would be tuberculous. This is favored by the fact that the lymphatics of the pericardium empty, in common with those of the pleura, either directly or *via* the glands at the base of the pericardium, into the glands about the large bronchi and the root of the aorta. The frequency with which tuberculous lesions affect these glands makes them the possible source of pericardial infection. To be sure, in thus explaining infection of the pericardium we must invoke a reversal of the normal current in the lymph vessels; but this phenomenon has been shown to occur by v. Recklinghausen,[3] and its results have been observed especially in the transmission of malignant neoplasms (*vide* article by Witte[4] on "Retrogressive Lymphatic Transportation of Carcinoma"). The choked condition of the lymph spaces in the tuberculous peribronchial glands would favor such a reversal of the direction of the lymph flow. Here, again, we would expect to find, at least frequently, miliary tubercles or their remains, as in vascular tuberculosis; but a lymphatic tuberculosis is likely to be a much more trivial affair, recoverable, and leading often to a simple sclerosis of the perilymphatic tissue. Hence it is possible that an adhesive pericarditis might result in this way, extensive or limited, and a few years after recovery no morphological evidences of its tuberculous nature remain. The connection of such a pericarditis with fibrous pleuritis is plain.

Direct infection of the pericardium by trauma can scarcely come under consideration in this matter of simple fibrous pericarditis. The occurrence of a fibrous mediastino-pericarditis due to repeated pressure on the sternum in certain trades, while affirmed by a few, is not generally credited. Roberts[5] states that he has met with no such cases. M. Labbé[6] reports a case of fatal cardiac incompetence following a pericarditis which he considered due to a blow on the chest. Such instances are extremely rare.

Of sources of direct extension the heart and the parts of the large vessels within the pericardium must be least common, since they are so rarely the seat of any extensive inflammatory processes. No relation between the simple diffuse chronic myocarditis and pericarditis could

be found in the cases studied, except in those cases in which the pericarditis, being primary, had affected the underlying myocardium. Localized myocarditis, acute or tuberculous, would leave sufficient evidences to determine the source of the secondary pericarditis. From the mediastinum infection might travel readily enough to and into the pericardium. Except in the lymph glands, infections in this space are rare, and a resulting process in the pericardium would have its course plainly indicated. But in the mediastinal lymph glands we have probably the commonest site of quiescent tuberculosis. These glands are in the closest relation to the pericardium possible, especially over its base. Another place of close contact is where the pericardium is carried along the left branch of the pulmonary artery, passing between the artery and the left bronchus. A gland seems to lie between this extension of the pericardium and the bronchus quite constantly. The small glands of the anterior mediastinum are also to be remembered, as they are often tuberculous. There can be no question of the relationship of these glands to tuberculosis of the pericardium. This relation has been consistently mentioned by every writer on tuberculous pericarditis, but the manner of infection has not been so clearly indicated. The possibility of dissemination of the bacilli by way of the lymphatics has already been considered. Occasionally a caseating gland breaks into the sac and erupts into this space, resulting in a rapid, caseating, or purulent inflammation of the entire surface. Such cases have been reported by Kast[7] and Mickle.[8] But generally the mechanism of the infection is not so evident. Usually it is simply found that a number of caseous glands lie upon the external surface of the pericardium, united to it by adhesions which sometimes are dense, but often only slight, and there is no continuity of the tuberculous lesions of the gland and those of the sac. In some cases the inflammation between the membranes is typically tuberculous, but in many it presents simply a mass of fibrous tissue of varying thickness, and neither macroscopically nor microscopically can a single feature warranting a diagnosis of tuberculosis be found. Such a case is the following;

Case I.—J. M. G., colored, male, aged thirty-two years. Died with a general anasarca and marked albuminuria, giving rise to the diagnosis of chronic parenchymatous nephritis. No history of rheumatism or other acute inflammatory process was obtained. At autopsy the diagnosis was sustained; but, in addition, an extensive tuberculosis was found, with caseous and ulcerative lesions in the lungs, intestinal ulcers, and caseous and miliary tubercles in the abdominal and mediastinal lymph glands. There was also found an extensive bilateral fibrous pleuritis and a gray thrombus in the right internal jugular vein. In the right auricular appendix was a small mixed thrombus infiltrated with leucocytes. Both layers of the pericardium were thickened about equally, each being perhaps 2 mm. thick, and uniting them throughout was a layer of soft, bluish-gray connective tissue, which was easily torn,

though firmer at the base than lower down. Nowhere could anything resembling tuberculous lesions be found in the pericardium or in the exudate. The glands at the base of the pericardium, particularly over the right auricle, were closely adherent to the external surface of the membrane; but a careful dissection of these glands, supplemented by microscopical examination of the intervening tissues between these glands and the pericardium failed to show any extension of the tuberculous lesions outside of the gland capsule. A piece of the pericardium, with the soft exudate, was placed in the abdominal cavity of a guinea-pig; the animal remained in good health, and, when killed, eighty days later, showed no tuberculous lesions. Sections were made through many parts of the pericardium and examined microscopically, always negatively as to any lesions resembling those of tuberculosis. Everywhere the structure was that of a poorly vascularized, new-formed connective tissue, with few cells, which nowhere showed any tendency toward grouping. A few sections were stained for tubercle bacilli, with negative results. On the contrary, the lymph glands immediately adjacent to the pericardium showed all the characteristics of tuberculosis.

This illustrates a case which is almost certainly tuberculous, the origin probably from the glands in contact with the pericardium, but in which the manner of extension to the inner surface of the sac cannot be found, and in which the resulting lesion bears none of the characteristic anatomical evidences of its tuberculous origin. This case is but one of a number. Of the twenty-four cases of total obliteration of unexplained etiology, tuberculous lesions of the glands immediately adjacent to the pericardium were noted in ten. Inability to locate any direct extension from the tuberculous glands to the pericardium was likewise the result in two cases of tuberculous pericarditis, with abundant characteristic nodular lesions, that I have examined.

From this it is evident that a condition of total or partial synechia of the pericardium may and often does come from tuberculous mediastinal glands, without the healed lesions of the pericardium bearing any traces of their tuberculous origin. Two possible explanations suggest themselves. One is that the changes in the pericardium, although due to infection by the specific organism, at no time presented the anatomical characteristics of such an infection. It is by no means rare for tuberculosis in other localities to fail to produce the usual structural changes, and this is particularly true of serous surfaces. A. N. Perón[9] has shown that in tuberculous pleurisy the lesions in the early stages are not different from those of a simple inflammation except for the presence of the bacillus, and even later distinctive tubercles often do not form, so that in the healed condition there is no evidence of tuberculosis. The second explanation is derived from a comparison with the conditions about tuberculous glands other than those of the mediastinum. It is well known that the diseased glands of a chain in the neck are frequently fused by dense masses of firm fibrous tissue. Microscopical examination of this shows it to consist simply of fibrous tissue,

poor in cells, and nowhere presenting any of the evidences of tuberculosis. The capsule of the glands is found to be outside of all characteristically tuberculous cellular groupings. On the other hand, this process of sclerosis may extend for considerable distances from the nearest tuberculous glands. Since the sclerosis is seen outside of glands that are still increasing in size, it cannot be considered as the results of a healed tuberculosis. A distinct resemblance between this process and that seen in the pericardium is evident. The explanation that is most reasonable, and one quite generally accepted, is that the periglandular fibrous hyperplasia is due to the action of the products of the tubercle bacilli that have diffused outward into the tissues. The sclerogenic action of tubercle toxin is marked, especially when it is so diluted or so composed that the necrogenic action is excluded. J. Auclair[10] claims to have been able to obtain from cultures of tubercle bacilli two different substances, one soluble in ether, the other in chloroform, the first of which produces the necrogenic effects, the other only the sclerogenic. It would seem quite reasonable to assume that a similar diffusion of sclerogenic substances occurring from the peribronchial and other mediastinal glands would invade the pericardium or pleura and lead to a simple connective-tissue formation in these cavities. In favor of this view is the fact that the two sacs and the mediastinal tissues are often found fused together by a mass of firm fibrous tissue enclosing tuberculous glands; and, further, that in many of the specimens of fibroplastic pericarditis the lesions are definitely older over the base—*i. e.*, in close proximity to the glands, than elsewhere, as shown in Case I.

The pleura is probably the most frequent source of non-tuberculous pericarditis, except for the rheumatic form, if we consider the frequency of pericarditis in pneumonia, which caused twenty-nine of the seventy-one acute cases found at autopsy. It is not to be denied that rheumatism is a more common cause clinically, but the rheumatic cases do not often come to autopsy in the acute stage. Pneumococcus pericarditis, while perhaps sometimes hæmatogenic, is oftenest produced by direct extension from the pleura. It is not uncommon to see the inflammatory process just beginning over the inner surface of the pericardium in closest relation to the inflamed pleura. Such a process, healing, might leave pleural and pericardial adhesions; undoubtedly it sometimes does. But it is universally admitted that tuberculosis is the most frequent cause of pleural inflammation, and it seems proper to assume that many of the pericardial adhesions result from the extension of this process through the pleuropericardial wall. This extension may be by growth of the tubercles through the wall, or perhaps by diffusion of the sclerogenic products of the organism. A roundabout route may also be assumed, the mediastinal glands becoming infected from the pleura, and in turn infecting the pericardium in the ways before mentioned.

Occasionally cases of tuberculous peritonitis are accompanied by tuberculous pericarditis, which seems due to an extension through the lymph vessels of the diaphragm. More often this condition is accompanied by tuberculosis of the pleura, sometimes also of the meninges and joints, seeming to be a matter of a general tuberculous serositis. Rarely a simple peritonitis produces inflammation of the pericardium. None of the cases in this series, however, gave evidence of relation to the peritoneum.

By the above process of exclusion it has been attempted to show that *many cases of adherent pericardium are probably of tuberculous origin, although not showing any anatomical characteristics of tuberculosis.* However, it is to be understood that this is not the only cause. Many other affections of the pericardium may heal and leave adhesions, and this may occur as well in an individual with tuberculous peribronchial glands as in anyone else, so the existence of caseous or calcified glands is not conclusive evidence that adhesions found in the pericardial sac are due to tuberculosis. However, when the patient's history shows freedom from rheumatism, pneumonia, and other acute processes, and autopsy reveals a pericardial synechia accompanied by a similar condition in the pleural cavity, or healed or active tuberculosis in the mediastinal glands, or both, the assumption that tuberculosis has caused the pericardial adhesions cannot often be wrong.

Returning to the forty-three cases of fibrous pericarditis in which no etiology has been assigned, it is found that forty showed pleuritis, and in eighteen healed tuberculosis was demonstrated. Of these eighteen it seems proper to consider tuberculosis the most probable cause of the pericarditis in thirteen, for the aforementioned reason. The following cases are cited to illustrate these points:

CASE II.—A negress, aged thirty-five years, who died of uræmia, was found to have, in addition to an advanced chronic nephritis, a total obliteration of the pericardial cavity by firm fibrous adhesions. Both pleural cavities were similarly obliterated. In the lungs were a few small areas of caseating tuberculosis near the apices, and a few miliary nodules were present in the spleen. There was a general tuberculous lymphadenitis, affecting principally the mesenteric, retroperitoneal, anterior mediastinal, and peribronchial glands. The glands about the pericardium were intimately adherent to it. No caseation, miliary nodules, or other evidences of tuberculosis were seen in the layers of the adhesions. The heart weighed 440 grammes, the enlargement being in the left ventricle, and explained by the renal disorder.

CASE III.—A man, aged twenty-four years; death from phthisis and amyloidosis of the abdominal viscera. The pericardium was adherent over the entire surface of the heart, the adhesions showing no caseous or other distinctly tuberculous alterations. Both pleural cavities were likewise obliterated, and the lungs were firmly bound to the external surface of the pericardium. Advanced ulcerative and nodular tuberculosis affected both lungs extensively. The peribronchial lymph glands

were much enlarged and contained tuberculous areas. It is recorded that the heart was "somewhat enlarged," but there are no details as to the changes.

CASE IV.—Man, aged sixty-six years; death from general tuberculosis of pulmonary origin. Both pleural cavities obliterated by fibrous adhesions, and the lungs thus bound to the pericardium. Uniting the heart to the pericardium beneath the adherent pleura is a band of fibrous tissue.

Of the twenty-two remaining cases of fibrous synechia in the series without assignable etiology there are fifteen accompanied by a fibrous pleuritis, without other anatomical changes being found to explain the pericardial adhesions. Of the other seven a ready explanation is found in four. One was due to extension of a sarcoma, two to extension of aortic aneurism, and one from a healed myocardial inflammation.

The cases accompanying fibrous pleuritis, and without indications of the cause of either the pleuritis or the pericarditis, are fourteen in number. Of these only the following are of special interest:

CASE V.—Man, aged thirty-eight years; died from asystole. The clinical diagnosis was: Chronic adhesive pericarditis; cardiac hypertrophy, with dilatation; pressure on recurrent laryngeal nerve, with resulting paralysis. At autopsy the obliteration of the pericardial cavity by adhesions was found to be complete, and externally it was firmly united to the pleura and to the diaphragm. The heart was hypertrophied, there was chronic mitral and tricuspid endocarditis, and both auricles and the right ventricle were dilated. General passive congestion, with anasarca, was present, and a renal infarct was found, the embolus arising in a thrombus in the left auricle. Both pleural cavities were obliterated by firm adhesions, and where the left recurrent laryngeal nerve passed beneath the aorta it was compressed for a distance of one-half an inch by dense fibrous tissue. No evidences of tuberculosis were found, and the history states that the patient had not had rheumatism or other acute infection. This case was reported by Dr. J. B. Herrick[11] before the Chicago Medical Society, February 9, 1898.

CASE VI.—Male, aged fifty-seven years; death from asystole, secondary to chronic interstitial nephritis, arterio-sclerosis, and myocarditis. A few thin, fibrous adhesions passed from the right ventricle to the pericardium. A few fibrous adhesions were present in the pleural cavities. No tuberculosis. In the left auricular appendix was a mixed thrombus.

CASE VII.—Man, aged sixty years; died from cardiac failure. The pericardial cavity was obliterated by firm, old adhesions. Adhesions were abundant and strong in the pleural cavities. No tuberculosis. Clinically, the case was diagnosed as chronic endocarditis, but at autopsy there were no changes in the valves, although the heart was hypertrophied and dilated.

CASE VIII.—Man, aged sixty-one years; death from an uncompensated aortic stenosis. Adhesions obliterated the pericardial cavity over the base of the heart. In the left auricle was a mixed thrombus. Both pleural cavities contained fibrous adhesions, and in the right apex were calcareous nodules.

There were four cases in which no explanation whatever of the finding was possible, there being no healed tuberculous or fibrous pleuritis, nor yet any history of any acute infectious process. There are, therefore, some eighteen cases of fibrous pericarditis in which it is impossible to find the cause, except that in all but four it was associated with more or less extensive similar changes in the pleural sacs. To attempt to state any one thing as the cause in any of these cases would be mere guesswork; but the frequency of the co-existence of pleuritis is, perhaps, of some significance. If we are to consider the cause of the two conditions as the same, the occurrence of fibrous pleuritis as the result of tuberculosis without recognizable lesions must be considered, and also the relation to previous pneumonia and simple acute pleuritis. Nephritis accompanied many of these cases, and its relation to fibrous pericarditis, although doubtful, is of interest. Serous inflammations in nephritis generally mark the termination of the chronic interstitial affection, and Bosc[12] seems to consider it as altogether a terminal occurrence. He states that it does not seem capable of regression, and is followed by death in some days, or, rather, it is present at the moment of death. On the other hand, W. Ewart[13] believes that effusion may occur transiently, without evidences of acute pericarditis, in Bright's disease, as well as in rheumatism and cardiac troubles; usually this is of mechanical origin, but, as in the case of analogous pleural effusions, they may be dependent upon a subacute inflammation. If Ewart's ideas are correct, then nephritic pericarditis may occasionally be recoverable, and adhesions may originate in this way. But allowing for uræmia, pneumonia, and all the evident sources of pericardial adhesion, there remain a few cases the origin of which can only be conjectured from the anatomical findings. In these cases the history is often of help, giving evidence of pneumonia, rheumatism, acute infections of other sorts, which offer suggestions, while not permitting a positive conclusion.

Rheumatic Pericarditis. Eight cases of fibrous synechia in this series were directly attributable to rheumatic pericarditis, the following being illustrative:

Case IX.—L. G., aged fifteen years, female; St. Elizabeth's Hospital, service of Dr. Sanger Brown, autopsy by Dr. Le Count. This girl entered the hospital with severe chorea and acute articular rheumatism. Death resulted from asystole. At autopsy general œdema and passive congestion of cardiac incompetence were marked. The heart was greatly enlarged, weighing 840 grammes with the adherent pericardium, and the cavities were much dilated. The mitral valves were incompetent, and the aortic cusps were the seat of a recent verrucous endocarditis. The pleural cavities contained no adhesions, and no traces of tuberculosis could be found either in the lungs or in the lymph glands. Although the peribronchial glands were enlarged and soft, they contained nothing that could be interpreted as tubercles. The pericardial sac was completely obliterated by firm adhesions, which

when torn left a reddish surface, sprinkled with small grayish and yellowish points, some of which protruded. This appearance was identical with that of an acute miliary tuberculosis. Microscopically, the "tubercles" were found to correspond to small foci of round cells embedded in the adhesions, which nowhere had the structure of tubercles, although many pieces from different parts of the heart were examined. A piece of this tissue placed in the abdominal cavity of a guinea-pig did not produce tuberculosis, and sections stained for tubercle bacilli did not reveal their presence. Sections of the lymph glands showed the enlargement to be due to a simple endothelial and connective-tissue hyperplasia, with no evidences of tuberculosis.

CASE X.—Man, aged twenty-five years; died with asystole. Five months before death this patient had an attack of acute articular rheumatism, and one month later the cardiac symptoms became manifest. The clinical diagnosis was chronic endocarditis, cardiac hypertrophy, and acute dilatation. On opening the thoracic cavity the pericardium was found to present an unusually large surface, extending 10 cm. to the left and 6 cm. to the right of the median line. On the left side there were a few adhesions between the lungs and the pericardium, but *there were no adhesions to the chest wall.* On opening the pericardium it was found that although the external layer was not noticeably thickened it was united to the epicardium by firm fibrous adhesions, separated with difficulty. The heart was much enlarged, weighing 700 grammes; the ventricles were dilated, the right being 11 cm. in depth, the left $10\frac{1}{2}$ cm. Both auriculo-ventricular orifices were much enlarged. The myocardium showed no macroscopical changes. All the valvular surfaces were normal. General anasarca and passive congestion of the lungs, liver, spleen, and kidneys gave evidence of the valvular incompetence. No evidences of tuberculosis could be found in the lungs, pleura, glands, or elsewhere. There were no pleural adhesions except those uniting the left lung to the pericardium, which presumably originated from the latter.

CASE XI.—A woman, aged twenty-two years; death from asystole, with the following clinical diagnosis: Mitral regurgitation and stenosis, dilatation of the heart, double hydrothorax, hydroperitoneum, thrombosis of the left axillary vein. Her trouble all dated to an attack of rheumatism five weeks before death, with evidences of involvement of the heart, although no positive evidences of pericarditis were obtained while she was in the hospital. Three years previously she had had an attack of articular rheumatism, not accompanied or followed by cardiac manifestations. The anatomical diagnosis was as follows: Acute mitral and aortic endocarditis; mitral stenosis; hypertrophy and dilatation of the heart; thrombosis of the left auricle and of the left subclavian and axillary veins; general anasarca; healed splenic infarct; passive congestion of the liver and spleen; fibrous internal and external pericarditis. In this case the external surface of the pericardium was found adherent to both lungs where they were in contact with it, and over the apex to the chest wall. The cavity was completely obliterated by very firm fibrous adhesions, showing no nodules, caseous or fibrous. No adhesions were found in the pleural cavity except those about the pericardium, and the glands and lungs showed no evidences of tuberculosis.

CASE XII.—Man, aged thirty-five years. Clinical diagnosis: Cerebral embolism, chronic degeneration of the kidneys, left heart hyper-

trophy, œdema of the lungs. Anatomical diagnosis: Chronic mitral, tricuspid, and aortic endocarditis; hypertrophy and dilatation of the heart; softening of the left basal ganglia and left cerebral cortex; renal infarct; general passive congestion; cirrhosis of the liver; chronic fibrous pericarditis; and localized fibrous pleuritis. On the outer surface the pericardium was attached to the left lung by one fibrous band; internally the cavity was totally obliterated by firm fibrous adhesions, simple in character. In this case cardiac symptoms were first observed during an attack of rheumatism four months before death, with dyspnœa, which had been marked up to the time of death, and by cerebral embolism.

Case XIII.—Woman, aged forty-five years, dying with asystole. Anatomical diagnosis as follows: Acute and chronic mitral and aortic endocarditis; mitral stenosis; thrombus in right auricular appendix; infarcts in the lungs, with localized fibrinous pleuritis; general passive congestion; obliterative fibrous pericarditis. The pericardial sac was obliterated by simple fibrous adhesions, and externally adherent to the right lung. No tuberculosis. This patient had repeated attacks of rheumatism during the last twenty years of her life. First noticed cardiac symptoms twelve weeks before death, after exposure to cold, which exposure resulted in no other trouble besides that of the heart.

Comparing these cases with those of tuberculous origin, the following anatomical differences are seen: *In the rheumatic there is a total absence of tuberculous lesions in the mediastinal glands and lungs; the pleuritis existing is generally localized at the pleuropericardial surfaces, seeming to be secondary to the pericarditis; endocardial changes are usually present, in all but one of those recorded; death is usually due to cardiac failure in the rheumatic, while this is the exception in the healed tuberculous pericarditis; the age of the subjects is, on the whole, younger in the rheumatic.* So far as the changes in the pericardium are concerned, they differ not at all from those of the simple fibroplastic changes sometimes produced by tuberculosis, as described previously. On the other hand, in one case (Case IX.) the changes resembled macroscopically so closely those of a miliary tuberculosis of the pericardium, with sclerotic changes predominating, that it required considerable microscopical and experimental evidence to exclude tuberculosis. It is of interest that all but the first of these eight cases was classified as of probable rheumatic origin on the autopsy findings alone, and the verification of this diagnosis by the history when subsequently investigated showed that the differences above described are sufficiently characteristic to be of value. The relative effects of rheumatic and tuberculous pericarditis on the heart itself, which are of much clinical interest, will be discussed later.

Calcific Pericarditis. Of this rather unusual form of pericardial lesion there are four examples found in this series.

Case XIV.—Man, aged thirty-five years; death from chronic nephritis. The pericardial change evidently attracted the attention of the examiner, for the clinical diagnosis made before autopsy reads: "Chronic nephritis; pleurisy, with effusion; pericarditis or medias-

tinal tumor." The anatomical findings were as follows: General anasarca; fibrous pleuritis and peritonitis; fibrous pericarditis, with calcareous plates; serofibrinous pleuritis; dilatation and hypertrophy of the heart; interstitial pneumonia; cirrhosis of the liver, with congestion of the spleen and gastro-intestinal tract; chronic diffuse nephritis. Externally the pericardium was bound firmly to the diaphragm and lungs; internally it was found obliterated completely, and embedded in the adhesions were large, thick, calcareous plates. No tuberculosis found anywhere.

CASE XV.—A "middle-aged" woman, who died after an operation for empyema. Unfortunately the record of this case is incomplete, it having been done at a private autopsy outside the hospital, and it is impossible to improve the data. The anatomical diagnosis reads: Right pleuritis and thoracotomy wound; left fibrinous pleuritis; adhesive fibrous pericarditis, with calcareous plates; atrophic cirrhosis, with passive congestion of the spleen. It is stated that the pericardial layers were adherent, and in the adhesions were found irregular, calcareous masses; one, on the anterior surface, measured 3 cm. by 2 cm.; on the left border was one one-half the size of the fist; over the diaphragmatic surface was one 2 cm. in diameter. The heart was the seat of some hypertrophy and more marked dilatation, the left ventricle measuring 9.5 cm. in depth. No tuberculous changes were found in the lungs. Here the pericardial change seems definitely older than in the pleura, for the latter is described as fibrinous.

CASE XVI.—A man, aged thirty-five years, who died from asystole, with the following clinical diagnosis: General arterio-sclerosis; chronic endocarditis; insufficiency of the mitral valves; chronic parenchymatous nephritis. The anatomical diagnosis: Chronic adhesive pericarditis, with calcification; chronic mitral endocarditis, with stenosis; hypertrophy and dilatation of the heart and tricuspid incompetence; chronic adhesive pleuritis; general passive congestion, with slight anasarca; multilobular cirrhosis; diffuse nephritis. The pericardial sac was completely obliterated; there were calcareous masses in the right and upper border and a large collection of "stony masses" in the sac proper. Externally it was adherent to the left lung. No tuberculosis found in the body.

CASE XVII.—Man, aged fifty-five years, who died of acute nephritis. At autopsy were found: Chronic fibrous pleuritis; chronic fibrous pericarditis, with calcification; atheroma of the aorta; acute parenchymatous nephritis; cirrhosis of the liver, with jaundice; acute myocarditis, with myocardial segmentation. The pericardial cavity was found completely obliterated by friable fibrous adhesions, greatly thickened, and between the layers of the pericardium toward the base was a large calcareous plate. There were no changes in the lungs, and traces of tuberculosis were not found.

Calcification of pericardial exudate is rare enough to be of considerable interest, although the occurrence of four cases in such a relatively small number of autopsies leads to the belief that its rarity lies chiefly in the infrequency with which it has been reported. In 1899 Fritz Diemer[14] was able to collect but ten cases, beside two of his own, in which the calcification was extensive, more or less completely covering

some part of the heart. Three of the four cases mentioned would come under this description. Perhaps the most remarkable instance of this condition was reported in this country in 1890 by Drummond.[15] In this case the greater part of the heart was surrounded by a stony covering; the right ventricle had to be opened with a saw. The calcification extended through the right auricle and through the entire thickness of the left ventricle. It varied from one-quarter to one inch in thickness, and seemed to involve the epicardium only, not the parietal layer, which could be stripped off.

The etiology of this condition is very obscure. Diemer states that in his twelve cases it was hinted at in six only. His series certainly throws no light on the subject, except in pointing out the fact that in only one was any tuberculous lesion found, and that not related to the pericardium. We know that calcification occurs only in dead or dying tissues, or inspissated exudates, which, from analogy, are probably purulent. Calcification of adhesions is seemingly the less common of the two. In all the four cases described in this series it seems most probable that these were instances of calcified inspissated exudates. What form of exudative pericarditis is most likely to heal and leave the proper conditions for calcification? Certainly not tuberculous, for a caseating tuberculosis of the pericardium would seldom last long enough for this, and if it did it would be accompanied by characteristic lesions locally or remotely; but in none of these four cases, and in only one of Diemer's twelve was tuberculosis found anywhere in the body. However, Püschmann[16] has reported a case of calcareous myocarditis of tuberculous origin. The purulent exudate which is least malignant and most prone to become sterile and inspissate is that arising in pneumococcus serositis, and, further, this is one of the most common causes of acute pericarditis. It also agrees with the conditions observed in most cases of calcareous pericardium, in usually leaving no traces of its origin in the lung except fibrous pleuritis. In the first case of this series the existence of a marked interstitial pneumonia is of significance, as also the pleural thickening in three of Diemer's cases. Case XV. gave a history of pneumonia eleven years before death, and also of attacks of rheumatism, the last two years before death. Unfortunately, in the other cases the history is incomplete. Ewart[17] has reported a case of extensive calcification of the pericardial adhesions resulting from rheumatism.

A most important as well as interesting observation is that in *each and every one of these four cases of calcareous pericarditis a marked cirrhosis of the liver was present.* On looking over Diemer's[14] cases it is found that in two of the twelve no reference as to the liver can be obtained. Of the remaining ten, in seven a distinct atrophic cirrhosis was observed; in one a fibrous perihepatitis, combined with advanced

syphilitic cirrhosis, was found; in another a fibrous perihepatitis alone; in but one was there no fibrous change in the liver. This striking feature seems to have been overlooked by Diemer. In only one of these nine was any reference made to alcoholism, this individual being rated as a moderate drinker; in one case—that of Drummond[15]—the macroscopical appearance of the liver suggested tuberculous cirrhosis, but this was not confirmed microscopically. In the remaining cases there is nothing to explain this cirrhosis. Further reference will be made to this matter later in considering the results of pericardial synechia.

Results of Pericardial Adhesions. For the most part, synechia of the pericardium seems to be altogether without effect on the patient, and it is not usually recognizable during life, even in the cases in which it does produce some effects that are readily referable to the heart or circulatory system. Like the fibrous pleuritis, it is essentially an autopsy finding. Of the fifty-seven cases of fibrous pericarditis, of whatever nature, in the entire series, in thirty-six of which the pericardial obliteration was total, the condition was recognized during life in but three. For the consideration of the effects of pericardial adhesions the cases may be conveniently divided into three classes:

1. Those with but one or a few bands of fibrous tissue connecting the parietal and visceral layers.

2. Complete or nearly complete obliteration of the pericardial sac, without adhesions between the external surface and the surrounding structures.

3. Pericardial sac completely obliterated and adherent externally to the adjacent structures—that is, combined internal and external pericarditis.

It is hardly probable that a few bands such as are often seen lying upon the auricles, or attaching the apex of the heart to the parietal pericardium, interfere in any appreciable degree with the action of the heart.

More extensive adhesion may, and in many cases most certainly does, prevent perfect cardiac function. It is quite generally recognized that in the third group of cases, in which the heart is bound to the pericardium, and this in turn to the chest wall, the lungs, the posterior mediastinum, and the diaphragm, that cardiac dilatation, hypertrophy, and ultimately incompetence often result. This process is seen particularly in rheumatic cases, and is one of the most rapidly fatal of the cardiac complications of rheumatism, if not the most so. From the nature of the changes present, an incompetence established in this way is absolutely unaffected by treatment, and the course is rapidly and progressively downward. Pericardial obliteration from causes other than rheumatism less often leads to cardiac failure. It will be recalled that in the cases of this series ascribed to tuberculosis there were no deaths

at all from asystole, whereas in those of rheumatic origin the cause of death lay in the heart alone, either from the pericardial lesion or from valvular changes, or both, in all but one, which terminated prematurely from cerebral embolism (Case XII.). English writers have considered this matter of asystole of pericardial origin most extensively, and ascribed to it the proper importance. The reason that the rheumatic pericarditis is so much more prone to cause fatal incompetence of the heart is ascribed by them quite generally to changes occurring in the myocardium during the rheumatism; in other words, rheumatic pericarditis is generally a pancarditis affecting valves, muscles, and pericardium together. From this condition dilatation results, and Dickenson[18] argues that the chief effect of the pericardial adhesion is to prevent this dilatation from being overcome as the heart gains strength—*i. e.*, the dilatation is primary, and not due to the adhesions. Hypertrophy follows, but under disadvantages, and compensation is never attained. "Thus it is," he says, "that adhesions following upon non-rheumatic pericarditis, which even in children is attended by little enfeeblement of the heart, proves generally harmless."

While not questioning in any way the logic of Dickenson's reasoning, nor doubting the correctness of the inference, there is another factor that does not seem to have been brought forward. This is based upon the function of the pericardium as determined by H. L. Barnard.[19] Barnard has shown that its function is, beyond that of providing a smooth surface to permit motion, to limit dilatation of the heart and to relieve strain upon the muscle, especially upon the right ventricle during diastole. He compares its function in relation to the heart muscle to that which the fibrous adventitia of the arteries bears to the muscular coat. First, he found that normal pericardium is practically inextensible. Then he demonstrated that, while the heart of a dog within the pericardial sac ruptured under a minimum pressure of 1¼ to 1¾ atmospheres, when freed from the pericardium it ruptured from a pressure of ¾ to 1 atmosphere. Rupture within the pericardium is preceded by a rupture of that sac, which gives way at its attachment to the great vessels. (Barnard says that this part of the pericardium seems to be the only part that contains nerves, and suggests that this may bear some relation to the subjective symptoms met with in pericardial effusions, heart dilatation, etc.) He further showed that a heart which within the pericardial sac could be made to take 10.5 c.c. of fluid, when removed from the sac dilated to hold an additional 16 c.c., showing the support of the pericardium to the heart in dilatation. In pericarditis with effusion, the alterations of the pericardial tissue by the fluid and by inflammatory changes permit it to yield under the pressure of the accumulating exudate. If no adhesions form without, it returns to its normal size as the fluid is absorbed; but when it is fixed to the

chest wall, the diaphragm, and other structures, as the fluid subsides it cannot return to its original dimensions, at least not entirely, and so the heart, whether inflamed or not, lacking any support from the outside, dilates as the fluid disappears until the two pericardial layers are again in contact. Furthermore, in a pericarditis with effusion, the heart and pericardium are separated by a considerable space over most of their surfaces. The heart cannot well become adherent until the fluid is absorbed, and if the pericardium is adherent externally adhesion between its two surfaces implies a dilatation of the heart to render this interpericardial adhesion possible. Now, rheumatic pericarditis is often a pericarditis with effusion, while the form of tuberculous pericarditis under consideration in this paper is presumably a simple plastic process, occurring as it does slowly and without symptoms. Hence the occurrence of cardiac incompetence often in one and seldom in the other. Myocardial degeneration and valvular lesions such as occur in rheumatism would, of course, favor the dilatation of the heart to a dangerous degree, from which it is ordinarily prevented by its own musculature. The chief difference between this theory and that of Dickenson is that in the latter the dilatation of the heart is considered primary; in the former it is considered as secondary to the distention of the pericardium.

Adhesions on the outer surface of the pericardium are most commonly thin filaments attaching it to the lung on either side, especially the left; such adhesions are of little importance, because of the elasticity of the lung. It is when the bands are firm and numerous, or the adhesion is of complete surfaces, and the pericardium is united to the chest wall, or to the lung, when it is in turn firmly united to the chest wall, or, perhaps most important of all, when the pericardium has spread over the diaphragm for a considerable distance, that the heart is affected. It is by no means unreasonable to consider that by the traction of these different points of attachment during inspiration, the diaphragm and the chest wall pulling in different directions, that a dilatation begun by the contraction of new-formed connective tissue would be increased. This traction during inspiration is accepted by many as the correct explanation of the pulsus paradoxus that is sometimes seen in the condition of pericarditis accompanied by mediastinitis. Of causes of simple plastic pericarditis with mediastinitis, other than rheumatism, pneumonia is the most prominent, for the inflammation, beginning outside of the pericardium, naturally leaves external adhesions. The simple plastic form of tuberculosis is probably seldom accompanied by much adhesion to the chest wall or diaphragm, unless occurring with a mediastinitis. J. H. Sequeira[30] has reported an interesting example of this condition occurring in an infant fifteen months old.

That adhesions limited to the pericardial cavity itself can so interfere with the action of the heart as to lead to asystole has been a much-

debated question. Many of the English writers are convinced that internal pericarditis alone, especially when of rheumatic origin, may do so. For example, A. G. Barrs[21] has reported four cases, all in young people, in which there were no adhesions to the anterior chest wall, and in one only a few adhesions to the lungs. Theo. Fischer,[22] writing on the importance of pericarditis in children, states that of a total of 196 cases of adherent pericarditis occurring in Guy's Hospital only eleven had adhesions to the anterior chest wall. As he found that in seven years' time, while there had been but four deaths due to valvular disease in children under fifteen years of age, there had been thirteen from adherent pericardium, it is evident that most of these fatal cases of adherent pericardium must have been without external pericarditis. Carl Hirsch[23] has made a valuable study of cardiac hypertrophy by ascertaining the ratio between the weight of the body and the weight of the heart and of its individual parts. He arrives at the conclusion that simple obliteration of the pericardium cannot bring about hypertrophy. This conclusion is, however, based on the findings in but six cases, without reference to the cause of the adhesions, nor to the existence of external adhesions. Dickenson[16] takes the same view, and states that in nearly all cases of rheumatic pericarditis there are adhesions externally, although in his own series of cases this condition was not marked in about half. However, it is to be remembered that in removing the sternum as ordinarily done at an autopsy, any abnormal adhesion of the pericardium anteriorly would be overlooked, as a rule, unless quite extensive.

An illustration of asystole, without external adhesions of consequence, is afforded by Case X. During life this was considered a case of broken compensation from valvular lesions. At autopsy the valves were found quite normal, the cavities dilated, walls hypertrophied, and the pericardial cavity was obliterated by strong adhesions without much thickening of the pericardial walls. There were a few adhesions between the left lung and the pericardium, but, although searched for, no other adhesions were found. Here, apparently, a fatal incompetence had resulted from simple internal adhesions. Macroscopically, the myocardium was unchanged.

How an internal obliterative pericarditis can interfere with the heart to any extent is difficult to see. Romberg[24] has suggested that the fibrous tissue, contracting, interferes with diastole, which supposition hardly agrees with the prominent dilatation in all these cases. Myocardial change in some instances is, perhaps, responsible, and this is sometimes to be distinguished only microscopically, as in a case reported by Jaccoud,[25] who considers that secondary changes are responsible for the incompetence of synechia more often than mere mechanical traction. In a number of cases that I have examined the myocardial lesions have

been slight and limited to the tissues immediately beneath the pericardium. This applies also to the fatty infiltration which is often seen to cut out the superficial part of the musculature. The mere inelasticity of the adherent pericardium is not sufficient to cause any significant interference with cardiac action, and "there is abundant evidence that the mere fact of obliteration of the pericardial cavity around a normal heart is a trivial impediment to its action, and excites neither hypertrophy nor dilatation." (Dickenson.[18]) The fatal termination of the process produced by the pericarditis is not generally long delayed; often it is within a year of the beginning of the acute inflammation. In the case of Jaccoud,[25] previously referred to, death occurred about three and a half months after the onset.

An interesting and important condition which results from pericardial synechia is that of "pericarditic pseudocirrhosis" of the liver. This was first described by F. Pick[26] in 1896, and has attracted much attention since that time. He stated that the cases resembled much the mixed form of cirrhosis of the liver, with enlargement of the liver, and ascites, but without jaundice, occurring especially early in life. In all these cases the pericarditis is latent. The cause of the symptoms he considered as eminently a cirrhosis due to connective tissue proliferation resulting from obstruction in the circulation of the liver, and to intense ascites through portal congestion. R. C. Cabot,[27] in 1898, reporting a case, collected seventeen others from the literature, and the reports have increased at a corresponding rate since that time. V. Eisenmenger[28] most correctly objects to the relation between passive congestion and cirrhosis assumed by Pick, for little hyperplasia of connective tissue results from passive congestion in the liver, and that which does form is in the centre of the lobule. The ascites, without œdema of the legs, he attributes to conditions outside the liver, chiefly torsion, compression, or angulation of the inferior vena cava by the pericardio-mediastinal adhesions; or else through a localized peritonitis, with some possible etiological relation to the pericarditis, at the transverse fissure of the liver. This last is, perhaps, most marked in the perihepatitis or "zuckergussleber" of Curschmann. One interesting observation is the following: In seven of Diemer's[14] ten cases of calcification of the pericardium, in which the condition of the liver was described, atrophic cirrhosis was present; in an eighth there was extensive perihepatitis and syphilitic cirrhosis; in the ninth there was perihepatitis alone; in one only of the ten there was no cirrhosis, but simple passive congestion. Only one gave a history of alcoholism, and that but moderate. In two of the three cases of pericarditis in Pick's first report the exudate was calcareous, and in both of these there was a slight but demonstrable cirrhosis of the liver; the third, in which the adhesions were not calcified, showed no cirrhosis. C. Bacaloglu[29]

reports a case of calcareous pericardium, not included among those of Diemer, showing a slight atrophic cirrhosis; no history given. In all four cases of calcification of the exudate in the writer's series a marked atrophic cirrhosis was present, although without ascites. Of these four, one gave a history of alcoholism (Case XVI.), but the other three were not alcoholics. From a study of the autopsy reports and history-sheets of these cases I am unable to ascribe any relationship between the pericarditis and the cirrhosis; but the coincidence is striking. However, it may be said that *while in fibrous pericarditis we do get a symptom-complex entitled to the name of pericarditic pseudocirrhosis, in calcareous pericarditis we frequently, in the great majority of reported cases, in fact, get a real atrophic cirrhosis.* This association of cirrhosis is not seen in cases of simple fibrous pericarditis. A case reported by Ewart,[17] of calcareous pericarditis, presented marked œdema and conditions leading to the diagnosis of atrophic cirrhosis, which did not exist. In this case the operation for establishment of collateral circulation by producing plastic peritonitis was performed.

An individual with both cardiac and tuberculous lesions may present them combined in an interesting form in the liver, as the so-called *cardiotuberculous liver.* This is simply a marked chronic passive congestion of the liver, plus tuberculous changes, which may be, according to Cousin,[30] either specific tuberculous lesions, or fibrous or fatty changes with or without tubercles. Tuberculous pericarditis offers the proper conditions for the development of this form of hepatitis, and it has been seen in many cases. Hutinel[31] has reported several cases in children, and considers the subject extensively.

W. H. Welch[32] has written of *thrombosis of the systemic veins* occurring in cardiac disease. In twenty-seven such cases collected by him the pericardium was found adherent three times, all being rheumatic. This has been observed in two of my series (I. and XI.); in both of these and in four others (V., VI., VIII., and XIII.) there were thrombi in the auricles. The snaring off or immobilization of parts of the auricles, particularly the appendix, furnishes most favorable conditions for thrombus formation, and embolism from this source is frequent. This is, therefore, a dangerous complication. Welch ascribes the thrombosis of the systemic veins in part to the eddying or whirling movement of the blood that may be set up in heart disease in the lower end of the jugulars and the adjacent parts of the innominate and subclavian veins, in part to the anatomical disposition and structure of the veins, but is inclined to think that infection is the important factor. In his first case, which resembled Case XI. of my series, in almost every detail, streptococci were found in the thrombus. In Case XI. the thrombus in the left axillary vein contained staphylococci, both aureus and albus, also the bacillus coli communis, and a small, strictly

anaërobic organism, the exact nature of which was indeterminate; it stained by Gram's method. From the heart's blood only S. pyogenes aureus and B. coli communis were obtained. The other case of thrombosis (Case I.) yielded S. pyogenes albus in cultures from the thrombus, which was not obtained in cultures from the heart's blood.

TERMINATIONS. It is quite probable that fibrous adhesions when once formed in the pericardial sac are not necessarily permanent. It is known that they are often removed from the peritoneal cavity in course of time, and this is probably true of the pericardial cavity. As the new-formed connective tissue becomes older and assumes its fibrillar structure the inevitable contraction that follows obliterates most of the bloodvessels, as in all scars. To this is added the incessant tugging of the heart, drawing them out and causing them to finally separate and form simple tags on the surface of the pericardium. This conception is formed from the frequent finding of the various stages of this process. As a rule, the parts of the adhesions that remain longest are those over the surface of the right auricle and also between the parietal layer and the apex and ventricular septum. Sibson[33] states that the reason for this is that these are the portions of the front of the heart which move least. As the "tugging" effect, as well as the interference with circulation by contraction, would be manifestly less in a completely obliterated sac than in isolated adhesions, the chances of complete absorption of adhesions will vary inversely with their extent, other conditions being equal. A healthy and strong heart would, of course, most readily free itself from the bands. Calcification is another possible termination.

The fibrous adhesions may also act as a "locus minores resistientiæ." Rubino[34] has shown the effect of injury to the pericardium on localization by first injuring the pericardium by heat, mechanically, and by severe cooling, and then injecting pus microbes into the general circulation. In this way he produced experimentally a pericarditis, which is the result that would be expected from what we know of the general laws of bacterial localization. That fibrous adhesions may produce in a slighter degree the same effect is not improbable, and it is not uncommon to see an acute pericardial inflammation engrafted upon a chronic adherent process in which the pericardium is but partly obliterated. On the other hand, total obliteration of the cavity would render secondary infection very unlikely to occur. McPhedran and Caven[35] in reporting a case of diffuse tuberculous hepatitis state their opinion that a coexisting pericarditis was implanted in this way on an older fibrous pericarditis.

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A CASE OF FIBROMA MOLLUSCUM.

By M. B. Hartzell, M.D.,
Instructor in Dermatology, University of Pennsylvania; Dermatologist to the Methodist Hospital of Philadelphia and to the Philadelphia Hospital.

In July of this year, G. J., a young, unmarried woman, aged twenty-six years, came to the Skin Dispensary of the University Hospital for advice concerning a disease of the skin characterized by the presence of a large number of variously sized tumors distributed over the entire cutaneous surface. These tumors were covered by normal skin, and varied in size from a hempseed to a pigeon's egg; they were soft and inelastic to the touch, the largest feeling like empty sacs, and, according as they were small or large, they were hemispherical and sessile, or pear-shaped and pedunculated. While all parts of the skin were affected, the tumors were most abundant and largest upon the arms. The history of the affection, very briefly, was that the tumors began to appear in infancy—it was asserted most positively by other members of the family that none were present at birth—and rapidly increased in number, new lesions continuing to appear up to the present time. Her general

health was in no way affected. The patient, while a physically well-developed woman, was evidently defective mentally.

Sections were made of a small tumor excised from the back, and examination of these showed that the epidermis was in no way altered, except that the interpapillary prolongations of the *rete mucosum* had to a large extent disappeared over the centre of the growth, owing to the flattening out of the underlying papillary layer of the corium. The

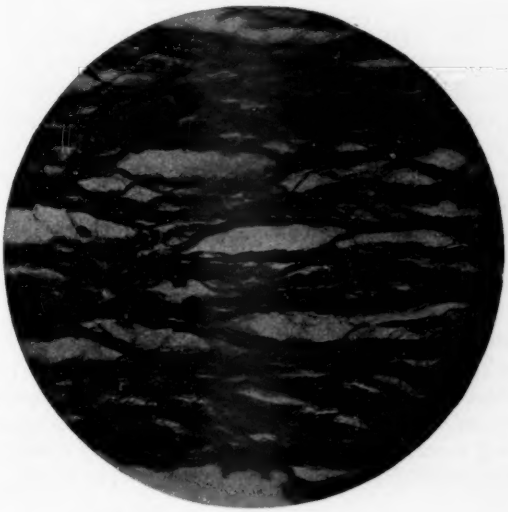
FIG. 1.

substance of the tumor consisted of fibrous tissue which at the periphery was arranged in a loose meshwork, while the central portion was quite compact. Many round and spindle-shaped deeply staining nuclei were observed, which were appreciably larger at the periphery of the growth than in the centre. With the employment of appropriate staining a considerable number of "mastzellen" could be seen. A few apparently normal hair-follicles and sebaceous glands, and a moderate number of small bloodvessels were present.

While not an extremely rare disease, fibroma molluscum is nevertheless quite uncommon, since, according to the statistics of the American Dermatological Association, it comprises but 0.090 per cent. of all diseases of the skin.

The etiology of the affection is extremely obscure, but heredity seems to play some part in its production, since it has been observed to occur in several members of the same family and in successive generations. Virchow has reported that a patient having fibroma molluscum stated that his grandfather, father, brothers, and sisters were likewise affected. The subjects of this form of tumor are often imperfectly developed physically and mentally, Hebra stating that all the cases observed by

FIG. 2.



him " were stunted in bodily growth and of more or less limited mental capacity." Later observations, however, have shown that there are numerous exceptions to this rule.

The number of tumors varies from a few to many hundreds. Mr. Hutchinson has reported a case, with a portrait, in which the face was the seat of hundreds of lesions, producing hideous deformity.

While there is a general agreement among those who have studied these tumors as to their anatomy, there is considerable diversity of opinion as to the part of the skin in which they have their origin. Rokitansky traced them to the deep part of the corium; Virchow believes that they originate in the connective-tissue framework of the

fat-lobules, and v. Recklinghausen asserts that they start from the fibrous sheath of the nerves. It is extremely likely that each one of these structures may serve as the starting-point for the fibrous hyperplasia, and that in some cases all these are involved.

Where the number of tumors is limited they may be removed by excision or the galvano-cautery, but when there are hundreds of them the treatment must be limited to the removal of the largest and most annoying.

HERPES ZOSTER AND ITS RELATION TO INTERNAL INFLAMMATIONS AND DISEASES, ESPECIALLY OF THE SEROUS MEMBRANES.

By Roland G. Curtin, M.D.,
of Philadelphia.

In September, 1890, I read a paper in the city of Denver, before the American Climatological Association, entitled "Is Herpes Zoster a Cause of Pleurisy and Peritonitis?" In that paper the histories of two cases were reported, one associated with pleurisy with effusion, and the other coming on with an attack of localized peritonitis. After reporting these cases I asked the following questions:

1. Was the internal disease a zoster eruption of the serous membrane?
2. Was the inflammation of the internal filaments of the nerves communicated to the pleura and peritoneum?
3. Did the internal inflammation cause the zoster?
4. Were both troubles independent and simply coincident?

I have since that time had the opportunity of observing other cases which may assist in settling at least two questions, and perhaps the third. I will repeat the two cases given in that paper, and then proceed to give other histories collected since which serve to bear upon this subject.

Case I. *Herpes zoster with pleurisy and effusion.* (Quoted from my former paper.)—Some time ago I had under my care a maiden lady who, in answer to the question "How old are you," replied, in a low voice, "To you I am sixty, but to other people I am fifty-six." She was in the last stage of locomotor ataxia, so that she could scarcely walk. While in this condition she was attacked with a short, intermittent, neuralgic pain in her side, at the left base of the chest. A careful examination failed to develop any physical signs of pleurisy. In time the pain was followed by a well-marked zoster, which satisfied me that the pain was preliminary to the herpetic eruption. About the time this eruption appeared I noticed a pain with every inspiration, like the "catching pain" of pleurisy, associated with considerable constitutional disturbance, and a dry, hacking cough. A little later my attention was called to the diminished movement of the

left side of the chest, which was almost immobile, and a careful physical examination revealed an effusion which half-filled the side of the chest. This effusion continued until the chest was entirely filled. In this case the zoster and the pleurisy seemed to be coincident.

CASE II. *Herpes zoster with dry pleurisy.* (Reported by Dr. H. H. Doan, Resident Physician of the Philadelphia Hospital.)—M. D., aged forty-two years, white, painter, single; weight, 120 pounds. Family history revealed no facts of any interest.

Previous History (November 29, 1891). Used whiskey to excess. Had initial lesion which, from history given, was evidently specific; malarial fever seven years ago; erysipelas twice; inflammatory rheumatism; friction rub heard on right side of chest five years ago; has been a patient in the drunkard's ward three times for delirium tremens. Two weeks ago he had a severe catching pain in the right side; it felt like a stitch. He never went to bed. Five days later a number of lesions appeared along the track of the seventh or eighth intercostal nerve of the right side, evidently an attack of "shingles" following pleurisy. Temperature was taken for one week. It reached 99.3° F. once. In this attack, which was an evident case of pleurisy at the seat of a former pleurisy, which he had five years before, it would be difficult to tell positively whether the pain of four weeks ago was from the affected nerves or the inflamed pleura.

CASE III. *Zoster and pleurisy.*—Prof. George H. Rohe, of Baltimore, having had his attention drawn to the subject under consideration by my first paper, in 1890, informed me that he had in his hospital a case of herpes zoster of the chest, with a marked pleuritic friction sound underneath the seat of the eruption. The friction sound was so well marked that he used the case to demonstrate the sounds of pleurisy to his students.

Dr. Rohe could not tell which was the initiatory affection in this case.

CASE IV. *Herpes zoster and pleurisy with chronic Bright's disease.* (Reported by Dr. P. Janney, Resident Physician, Philadelphia Hospital.)—Bridget McC., white, female, aged sixty years, born in Ireland, housewife, married.

Family History. Father and mother died of old age. One brother died at the age of seventeen of "enlargement of the liver." Four sisters and one brother living and healthy.

Previous History. Healthy, as a child. In 1845 had influenza with the rest of her family. In 1848 had typhus fever, and was sick three or four months. In 1856 had jaundice for three months. No history of biliary colic. Has had one living child and three miscarriages. No venereal history. Five years ago her feet became swollen, had pains in back, passed frequently small quantities of urine. Ever since has had more or less swelling of feet. Admitted to the Philadelphia Hospital about two years ago with diagnosis of Bright's disease.

History of Present Disease. In the middle of October, 1891, she felt a catch at intervals in the right side in the nipple line. October 30, 1891, had a constant severe pain in the seventh and eighth interspaces on the right side, and a friction rub was heard on November 2d. Chest was examined and a herpetic eruption was found in the seventh and eighth interspaces, extending around almost to the sternum and over

the seventh and eighth dorsal vertebræ. There was constant pain and a slight moist crackle heard on inspiration and expiration.

Examination of Urine. Urine passed in large quantities; pale and of low specific gravity, containing a slight trace of albumin, but no casts; acid in reaction. All physical signs disappeared in two weeks, showing that the attack of pleurisy was acute. The pleurisy in this case seemingly preceded the external eruption.

Case V. *Herpes zoster complicating phthisis and chronic Bright's disease.* (Reported by Dr. H. H. Doan, Resident Physician, Philadelphia Hospital.)—J. H. Diagnosis, phthisis and Bright's disease. A native of Ireland, tailor by occupation, single, tall and thin.

Family History. Mother asthmatic for many years. Father died of Bright's disease. No tuberculous history.

Health usually good; drank very heavily and used tobacco to excess. No venereal history. Smallpox in 1866.

Had cough for two years, losing flesh; expectoration very slight until May, 1891. In January, 1891, had a continued attack of diarrhœa. July 20th, noticed eruption on left side of the upper part of the abdomen; no pain. Before this, although following the appearance of the eruption, there was severe pain, lasting two weeks. At one time during this attack he had slight hæmoptysis, lasting for one day. Lost appetite; bowels constipated. On admission to hospital there was an herpetic eruption extending from spine at junction of fourth rib around to the nipple, with points of tenderness. No friction sounds heard, nor could fluid be demonstrated. Since admission he has had frequent chills, occurring at irregular intervals, with decided irregularities of temperature. No plasmodium found in blood.

Urine, acid and albuminous, with many granular and hyaline casts. Numerous bacilli found in sputum.

With tubercular phthisis we have more or less plastic pleurisy, and many observers have reported cases of zoster complicating tubercular disease of the lungs, and in their cases the pleurisy antedated the eruption.

Case VI. *Herpes zoster with localized peritonitis.* (Quoted from my former paper.)—I had a case which served to bear upon the subject under discussion—apparently an inflammation occurring in another serous membrane simultaneously with zoster.

A woman, aged thirty-five years, who was about four months pregnant, had what I had diagnosticated as a localized peritonitis, occurring on the right side of the upper part of the abdomen, just below the margin of the liver. There appeared later at the same position a well-marked, clustered eruption. At about the same time a deep-seated soreness and tenderness appeared, extending to the median line. The febrile symptoms were marked; respiration and movement caused a great increase in the pain, which was constant; and pain was caused by the flatus which was present passing through the bowels. She barely escaped aborting. Later on the uterus increased in size and caused much pain, which was relieved, but not entirely so, when she was delivered. It was some months before the soreness entirely disappeared. The diseases seemed to be coincident in this patient.

Case VII. *Herpes zoster following catarrhal appendicitis.*—M. L. had a sharp attack of catarrhal appendicitis with high fever, which ran its course in about seven days. The next week a sharp, lancinating pain

appeared, followed later by a zoster eruption on the right side of the abdomen, the right hip, and the upper part of the thigh. The localized peritonitis preceded the skin affection in this case.

CASE VIII. *Herpes zoster following arthritis and effusion of right knee.*—Judge A. Right knee-joint was much swollen and painful from rheumatic arthritis. This condition had continued for several weeks when an eruption of herpes zoster came on the outside of the knee and popliteal space, and ran the usual course of that affection. The eruption did not encircle the knee.

Here we find a case of hydro-arthritis of the knee-joint of six weeks' standing, the skin around it being secondarily affected by zoster.

CASE IX. *Influenza; meningitis; pleuropneumonia followed by herpes zoster.*—F. L. had an attack of influenza followed by meningitis and later on by a pleuropneumonia of the right lung. Late in convalescence he had a marked attack of herpes zoster situated on the right side of the lower anterior chest and upper part of the abdomen.

CASE X. *Hæmaturia and zoster.*—S. A. had, in May, 1899, an eruption of well-marked zoster over and above the position of the left kidney, extending around to the umbilicus and below it. In the third week of August, two and a half months later, he began to pass blood in his urine. This continued for months. There was no pain or other discomfort over the kidney or bladder except when he passed a clot, and then he felt a slight pain and discomfort at the symphysis pubis.

The quantity of blood constantly varied and continued for months. The color of the blood was generally dark or quite black. He had no constitutional disturbances. Two years ago, September, 1897, he had a similar eruption on his face, which lasted about ten days or two weeks; at the same time he told the doctor that his urine was high colored. The doctor examined it and said it was blood. This hemorrhage lasted four or five weeks. He never had any eruption or hæmaturia at any time other than these two occasions.

RECAPITULATION. 1. In Cases I., II., III., and VI. the herpes zoster seemed to come on at the same time as the internal inflammation.

2. In Cases IV., VII., VIII., and IX. the affection of the serous membrane preceded the eruption.

3. In Case V. the zoster appeared during the course of a chronic pleurisy of tubercular phthisis.

4. This résumé seems to indicate that inflammation of the serous membrane precedes the zoster; therefore, we may reason that the inflammation caused the eruption.

5. These conditions occur together or follow one another too frequently to be simply coincident.

I feel sure that the cases here reported settle at least some of the problems that I suggested in my former paper:

1. Was the internal disease a zoster eruption of the serous membrane?

2. Was the inflammation of the internal filaments of the nerve communicated to the pleura and peritoneum?

3. Did the internal inflammation cause the zoster?

4. Were both troubles independent and simply coincident?

While on this subject of zoster and internal diseases I will take the opportunity to state that I had a case, in 1874, of œsophageal cancer that had, late in the disease and over its seat, a complicating zoster eruption encircling the left side of the chest at the margin of the ribs. The eruption, it seemed to me, might be caused by the cancer, as the œsophagus is located to the left of the border of the vertebræ, near the roots of the nerves.

In 1890 to 1893 I had quite a number of cases of influenza and catarrhal fever complicated with zoster, generally coming on with the less acute influenzal attacks. A number of my friends reported to me one or more attacks of the same kind in their cases of acute and subacute influenza.

The subject being, I believe, a new one, I shall feel repaid if the attention of future observers is called to this interesting condition.

THE CHANGES OCCURRING IN STRIPED MUSCLE IN THE NEIGHBORHOOD OF MALIGNANT TUMORS.[1]

By Frank P. Anzinger, A.M., M.D.,
of Ann Arbor, Mich.

By far the greater part of the investigations made during the last decade into the nature of malignant tumors have been along the lines of their supposed parasitic origin. Yet, with all of the immense effort and labor extended in this direction, we are to-day not one step nearer the solution of the problem of the etiology of these growths. There is no sufficient evidence to prove that the peculiar bodies found in carcinoma cells are parasites, either bacterial, protozoic or blastomycetic, each of which they have from time to time been confidently proclaimed to be. It must be acknowledged that we are no nearer the demonstration of this problem than we were ten years ago, and the oft-repeated failures should convince us that if malignant neoplasms are parasitic in origin we must arrive at the discovery of the parasite by following lines different from those hitherto pursued. In the meantime, while this field of research is being so largely worked, other aspects of the tumor problem are being more or less neglected, particularly those along chemical lines.

Very little work has been done with the comparative chemistry of tumor cells and cells of normal tissues, or with the chemistry of the products of tumor-cells. The changes occurring in the cells in the

[1] From the Pathological Laboratory of the University of Michigan, Ann Arbor, Mich.

neighborhood of malignant tumors have also received but scanty attention.

In 1867, Otto Weber made observations on cancer in the pectoral and sternomastoid muscles. He described the manner of growth of carcinoma into the muscle, the presence of cancer-cells in the muscle-fibres, and the destruction of the muscle as the result of suppuration, but the finer structural changes were not observed. As a result of his study he concluded that cancer-cells were derived from muscle-elements. In 1870, Volkmann described the appearances of cancer in the pectoral muscle, the manner of invasion of the muscle by cancer-cells, the formation of cancer nodules, and a peculiar change in the muscle protoplasm, to which he applied the term *lacunar erosion*. He pointed out the fact that this change was due to the pressure of cancer-cells, and concluded that carcinoma cells invade muscle, but do not originate from muscle-elements.

The first careful study of the minute changes occurring in striped muscle in the neighborhood of malignant growths was made by Schaeffer in 1886. The material examined by him consisted of 3 cases of epithelioma of the lip, 2 cases of carcinoma of the pectoral muscle, 3 cases of carcinoma of the tongue, and 4 cases of sarcoma. The manner of invasion of the muscle and the gross appearances of muscle changes are first described by him, then the finer changes as produced both by sarcoma and carcinoma. The special changes observed were: Distortion of the fibres, atrophy, cloudy swelling, fatty degeneration, waxy change, fissuring, changes in staining, nuclear proliferation, hypertrophy, small-celled infiltration, and increase of the perimysium. He compared these changes with those found in other conditions, and concluded that the changes produced by tumors were not characteristic, but could be produced by other pathological agencies.

Since the work carried out by these three observers represented the sum total that had been done in this direction, it seemed worth while to extend this research with a view to settling several questions left untouched by them, particularly with reference to a comparison of the changes produced by sarcoma and carcinoma. While this work was in progress there appeared in April, 1900, an article upon the same subject by Fujinami. As this is the last and most important contribution in this line, I give his conclusions somewhat at length. These are based upon the study of 20 cases of sarcoma and 17 of carcinoma infiltrating muscle. The manner of invasion and the changes occurring in the contractile substance and muscle nuclei, and the fate of the primitive muscle bundle, are considered in detail. The results of this study are as follows:

1. The invasion of the muscle takes place in different ways, both through the lymph-spaces, lymph- and bloodvessels, and the sarcolemma.

The invasion of the sarcolemma by tumor-cells occurs most frequently in the case of carcinoma, and is especially pronounced when the direction of extension of the tumor is parallel with the axis of the muscle-bundle. It is seldom seen in sarcoma, occurring only in the case of the small round-cell variety. The penetration of the sarcolemma is dependent upon the biological relations of the tumor-cells, their direction of growth, and the variety of change in the muscle-substance.

2. The changes in the primitive muscle-bundle are of manifold variety, all possible changes occurring. Simple atrophy is the most frequent; and the most interesting are the nuclear proliferation, "ampullar atrophy" and formation of giant-cells. Those changes formerly regarded as regenerative in nature are to be regarded as being essentially degenerative, the primitive muscle-bundle in a certain stage of retrogression assuming relations similar to those at the beginning of its development.

3. The muscle-fibres show varied and irregular changes in thickness and length.

4. The changes produced in the primitive muscle-bundle by tumor-cells cannot be referred alone to mechanical pressure, but the changed biological relations of these cells must also produce chemical effects.

5. In the interstitial connective tissue there is often a reaction, as shown by leucocyte infiltration, fibroblastic proliferation, and proliferation of the intima of the vessels. These changes may also secondarily affect the primitive muscle-bundles. The infiltration of the perimysium is usually much more marked in the periphery of carcinoma than of sarcoma.

6. Cell-forms (sarcolytes, sarcoplasts) arise from the changed muscle-bundles, and under certain conditions it is probable that these take part in the formation of tumor-cells.

7. A connective-tissue-like change of the muscle-fibres is assumed.

8. The reaction of muscle to the invasion of a malignant tumor may be very varied according to the local conditions. The muscle does not play a passive rôle simply.

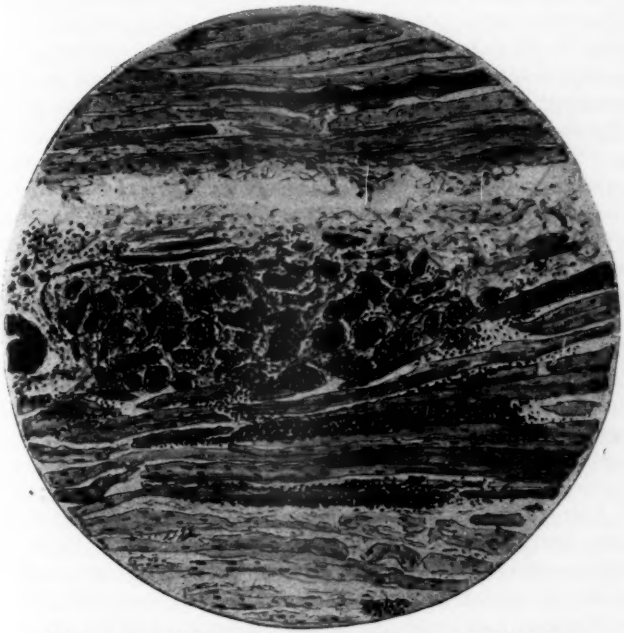
9. The fact that the muscle-fibres possess a characteristic appearance and show various changes is of practical value in the diagnosis of tumors, in so far as it aids in establishing the biological properties of the tumor-cells and their direction of growth.

10. There is no absolute difference between the muscle changes produced by sarcoma and carcinoma.

As the work begun by me before the appearance of Fujinami's paper had led me to conclusions differing from some of those of his, it was continued with renewed interest, and the results are given below. For the original suggestion of the work I am indebted to Dr. Warthin, who gave me access to the material of his laboratory, and my research has,

from time to time, been controlled by him. The material examined consisted of 30 cases of cancer of the breast infiltrating the pectoral muscle, 15 cases of epithelioma of the lip, 1 of cancer of the tongue, 1 case of lupus-carcinoma of the cheek, 2 secondary carcinomas of the diaphragm, 2 cases of sarcoma of the pectoral, 2 fibrosarcomas of the rectus abdominis, 2 sarcomas of the inferior maxilla, 1 sarcoma of forearm, 3 of femur, 1 of tibia, and 1 of general sarcomatosis of the skin. Examinations were made both of hardened and fresh material. Alcohol,

FIG. 1.



Carcinomatous nodule in pectoral muscle, showing manner of invasion. (Leitz obj. 3, eye-piece III.)

mercuric chloride, Flemming's, Müller's, and Zenker's fluids were used as fixing agents. The muscle was cut both transversely and longitudinally. Celloidin and paraffin embedding were both employed; and the following stains were used: hæmatoxylin and eosin, Van Gieson, carmines, polychrome, methylene blue, orcein, Russell method for cell-inclusions, Weigert nerve method, Mallory's reticulum method, and Plimmer's method.

MANNER OF INVASION. 1. *Carcinoma in Pectoral Muscle.* The primary carcinoma of the mammary gland advances by direct expan-

sion until the fascia of the pectoral muscle is reached. The latter serves as a barrier to further advance, and the carcinoma usually reaches the muscle through the lymphatics. Uninvolved fascia may separate the main mass of the tumor from the muscle when in the latter there are metastatic foci of tumor-cells in the lymph vessels about the bloodvessels. Serial sections show that these are often not metastases, but direct extensions of the growth through the vessels. When the nodules in the muscle reach a large size the muscle-fascia may be destroyed, but in the earlier stages it is rarely involved. Its normal appearance is, however, no evidence that there are no tumor nodules in the muscle beyond it. The muscle may be strewn with nodules varying in size from a pin-point to a walnut. They resemble the primary in structure. Arising in the interstitial tissue, they push the muscle-fibres apart and produce in it the most varied pathological changes. As the nodules increase in size there is more or less complete destruction of the fibres. Remains of these may be found in the cancer nodule. In infiltration with scirrhous carcinoma there is a marked proliferation of the perimysium, and this leads to the production of marked changes in the primitive bundle. Small islands of muscle-protoplasm and small groups of cancer-cells are surrounded by a dense mass of connective tissue. Invasion by way of the bloodvessels is rare. When the muscle is once reached further extension takes place through the lymph-spaces of the interstitial tissue and also through the sarcolemmar tubes.

2. *Epithelioma of the Lip.* The invasion of muscle is favored in this region by the structure of the muscle-fibres, these being short and irregular and the perimysium of a very loose texture. The irregular columns of epithelial cells fill in the meshwork of the perimysium; the younger cancer-cells infiltrate between the separate muscle-fibres and destroy these as they increase in size. Leucocyte infiltration is usually marked, and it is difficult to separate the leucocytes from the young cancer-cells. Cells undergoing hyaline change or cornification may also be mistaken for remnants of muscle. Small fragments of muscle-plasma and nuclei may sometimes be found in the central portion of a cancer nest; this appearance is, however, explained by the plane of the section, and is not due to an encapsulation of muscle by cancer-cells as has been supposed. Penetration of the sarcolemmar tubes by the young cancer-cells also occurs, but is more rare than in the case of breast cancer.

3. The invasion of muscle by secondary carcinoma in other regions is similar to that in these two places, the difference depending upon the local conditions in the muscle and the character of the tumor-cells.

4. *Sarcoma.* The invasion of muscle by sarcoma takes place by direct extension in the form of outgrowths of the main mass penetrating the muscle-bundles, extension of slender cords of cells between the

muscle-fibres, and also by lymphogenous and hæmatogenous metastasis. There is always much less reaction in the perimysium of muscle invaded by sarcoma than there is in the case of carcinoma. The ingrowths of sarcoma replace the perimysium, at first separating the muscle-bundles, which may show but slight change. As the proliferation of the tumor progresses there is gradual atrophy and disappearance of the muscle-fibres. These may, however, persist for a long time, and not infrequently small bits of muscle may be found scattered throughout the tumor. Many of these may appear as regeneration forms. In the case of universal sarcomatosis of the skin, large subcutaneous nodules were present which resembled greatly enlarged lymph-glands. On section these were found to contain in their central portion the remains of muscle-fibres. Round-cell sarcoma produces a more rapid destruction of muscle than the spindle-cell variety. On the whole the effects of the invasion of muscle by sarcoma appear to be chiefly mechanical, as the surviving bits of fibres usually show but little change in structure. The rapidity of growth in some cases causes a rapid disappearance of the muscle without preceding retrograde changes.

Changes in Muscle Produced by Invading Malignant Tumor. 1. *Atrophy.* This is the most frequent and constant change. It may be either mechanical or chemical in nature. In the case of scirrhous carcinoma, epithelioma, and sarcoma, the atrophy is limited to the area of muscle invaded by the growth, and is chiefly the result of pressure. In the case of non-scirrhous cancers of the breast, the atrophy may extend into the pectoral a long distance from the advance cells of the tumor, and may affect large bundles of muscle in which no cancer-cell is present. This cannot be explained as mechanical in origin; on the other hand, it is probable that it is due to disturbed biological or chemical relations. Both forms of atrophy may be present in the same case, but the chemical form is more characteristic of carcinoma. In scirrhous carcinoma, epithelioma, and sarcoma the length of the muscle-fibre is very frequently preserved, while its thickness is reduced to that of a mere thread. In the rapidly growing forms of breast cancer the fibres of the pectoral show great variation also in length.

2. *Hypertrophy.* The occurrence of hypertrophic fibres in the neighborhood of malignant growths was maintained by Schaeffer, who made measurements of the fibres in 2 cases of fibrosarcoma. But even in the case of actual measurements it is hardly possible to make such a diagnosis, inasmuch as the standard of size shows such individual differences. Large fibres occur very frequently among the atrophic ones, but this enlargement in the majority of cases is only relative. The size of the fibre is also increased in certain forms of degeneration, notably cloudy swelling.

3. *Changes in Morphology.* Very marked and varied changes in the morphology of the fibres occur. The parallel arrangement of the fibres

may be changed; they may be bent at various angles, often assuming a spiral form. The sarcolemma may be retracted from the muscle-substance, the latter being formed into wavy bands or broken up into scroll-like figures.

4. *Changes in Muscle-substance.* (*a*) *Alteration in the Striation.* In the atrophic fibres the cross striation is usually very sharply defined, the alternating light and dark bands being very narrow. Very frequently there is a longitudinal fibrillation with loss of the cross striation. The plasma is divided up into its fibrillar elements, and often at the cut end of a fibre these may be seen protruding from the sarcolemma in a brush-like manner. Except in the case of cloudy swelling and Zenker's necrosis, the longitudinal striation persists as long as the plasma remains intact.

(*b*) *Vacuolization.* This is a frequent change, and in some cases may be the only one present. It occurs often as a very early sign of degeneration. The vacuoles appear as clear or cloudy spaces in the plasma, which contrast strongly with the surrounding stained plasma. They may be sharply outlined as if punched out, or their border may be irregular, in many cases appearing as if fringed. They vary much in size and shape, and at times communicate by delicate grooves or channels. When located at the periphery of the fibre the sarcolemma may bridge over the vacuole or become invaginated into it. In the majority of cases they are to be interpreted as due either to hydropic degeneration or Zenker's necrosis.

(*c*) *Fissuring.* Fragmentation of the fibre may occur in any plane, transverse, longitudinal, or irregular. These changes may be partly of the nature of an artefact, but are undoubtedly pathological to a large extent, as they occur in the most carefully fixed specimens.

(*d*) *Waxy Degeneration.* Zenker's necrosis is of very common occurrence, particularly in the case of mammary cancer invasion of the pectorals, where it very often occurs at some distance from the tumor. Schaeffer maintained that this form of necrosis was not common near tumors, but in my cases it has been found both of frequent and extensive occurrence.

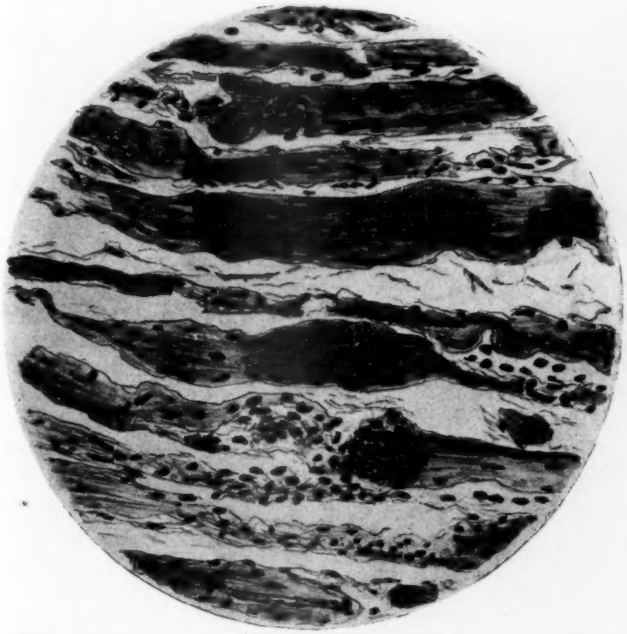
(*e*) *Cloudy Swelling.* This is of very frequent occurrence. The fibres are swollen, granular, stain heavily, and show a tendency to take both nuclear and diffuse stain (polychromatophilia).

(*f*) *Fatty Degeneration.* No fatty change was found in muscle in the vicinity of tumors, though repeated search was made with the use of osmic acid. Weber and Schaeffer state that it is of common occurrence under these circumstances, but Fujinami found only traces of it in the examination of fresh material.

Lacunar Erosion. The appearances described by Volkmann under the head of "lacunar erosion" occur not infrequently in invading

carcinoma of the pectoral. Muscle-fibres which otherwise may appear normal present one or more cup-shaped indentations of the plasma at some point along its margin. These depressions usually contain eight to twelve nuclei, which are small and irregular in outline, but from general appearances must be considered to be muscle nuclei. Other fibres may be found in which the muscle-substance is intact except at one or both poles, where it is interrupted by a concavity which may be shallow or deep. In these depressions are numerous deeply staining

FIG. 2.



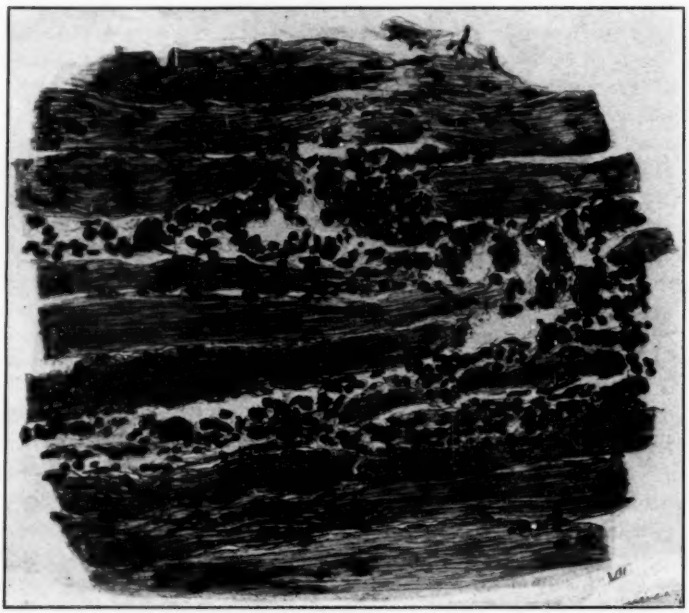
Pectoral muscle near periphery of mammary carcinoma, showing lacunar erosion and ampullar atrophy. (Leitz VII., eye-piece III.)

nuclei packed so closely that, with the low power, their character cannot be made out. Examination with high powers shows that the sarcolemma is prolonged for some distance beyond the muscle-substance, the two lines representing the cross section of the sarcolemma finally converge and meet. These apparent erosions are, therefore, explainable as being portions of the fibre where the muscle-substance has entirely disappeared as the result of degeneration, while the muscle nuclei have undergone proliferation. Other fibres show earlier stages of this process; beneath the sarcolemma the remains of the muscle-substance,

in the shape of granular detritus tinged with diffusely staining chromatin, are found. These apparent erosions are in the majority of cases not due to the presence of tumor-cells, but in some instances a group of the latter is found pushing the sarcolemma into the depression and presenting the appearance which led Volkmann to believe that the erosions were due to an "eating-out" action on the part of the tumor-cells.

5. *Changes in the Muscle Nuclei.* (*a*) *Proliferation.* This is a very common change, and presents many features of unusual interest.

FIG. 3.



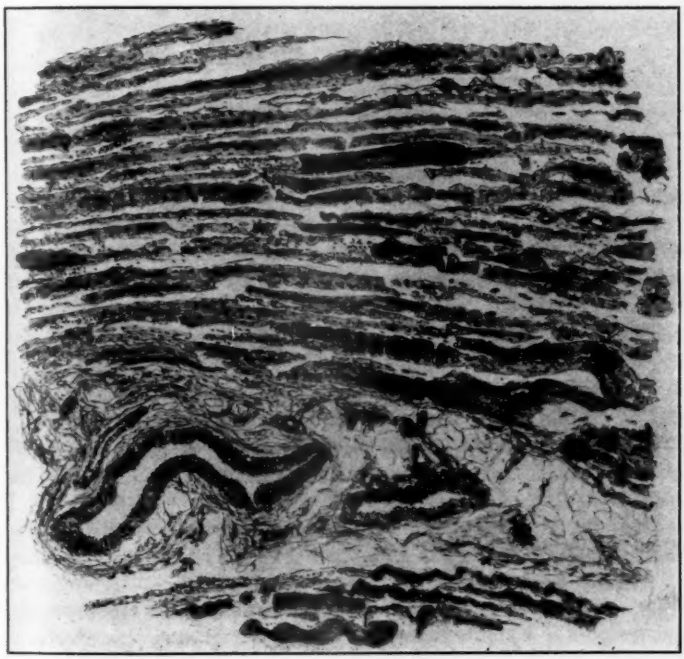
Invasion of pectoral muscle by mammary cancer, showing changes in muscle-substance and nuclei. (Leitz VII., eye-piece III. Camera lucida, reduced 1/3.)

Increase in the number of the nuclei leads to the formation of long nuclear chains, often sixty nuclei being counted in one chain. At other times they may be so numerous as to form a solid cord. In transverse sections the nuclei are often seen arranged in clumps replacing a portion of the plasma just beneath the sarcolemma. The central portion of the fibre may contain many nuclei. The collections of nuclei found in the cup-like erosions of the muscle-substance beneath the sarcolemma are undoubtedly muscle nuclei which have multiplied at these

points. Waldeyer has described appearances in diseased muscles in which the protoplasm is entirely replaced by muscle nuclei (Muskelschlaüche). In a few instances these were found by me, but were not so typical as suggested by some observers. No mitotic figures were found in any of my specimens, the proliferation appearing to be by direct division.

(*b*) *Changes in Morphology.* The normal muscle nucleus is oval in shape, and the average measurement is 7 to 11 μ in length and

FIG. 4.



Changes in pectoral muscle at some distance from edge of carcinoma. (Leitz III., eye-piece III. Camera lucida, reduced 1/3.)

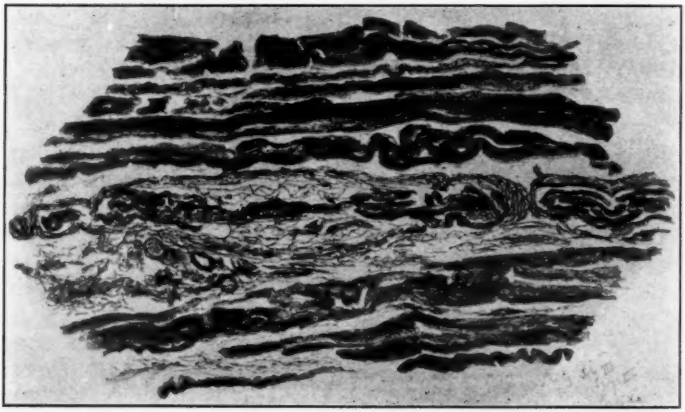
3 to 4 μ in breadth. Near malignant tumors they become very much elongated, some nuclei measuring 40 μ in length. Others are very short, some atrophic, and others hypertrophic. They may be very much altered in shape and staining power. The nuclei survive the destruction of the muscle-substance, but ultimately disappear also.

6. *Changes in the Sarcolemma.* The behavior of the sarcolemma during the destruction of the muscle protoplasm has already been men-

tioned. Its final fate is degeneration and complete disappearance, though some investigators believe that it may take part in the proliferation of the perimysium. After the disappearance of the muscle-substance the sarcolemmar tube may collapse or it may be entered by leucocytes and tumor-cells. Not infrequently it appears to have been distended by fluid.

7. *Changes in the Perimysium.* In infiltration of the perimysium with cancer-cells the connective tissue takes part in the formation of the stroma of the cancer nodule. The proliferation of the perimysium takes place, however, to a much greater extent in the case of spindle-

FIG. 5.



Muscle spindle in pectoral muscle near invading carcinoma, showing nuclear proliferation in intrafusal fibres near equator. Neighboring muscle shows retrograde changes. (Leitz III., eye-piece III. Camera lucida, reduced 1/3.)

cell and fibrosarcomas, epithelioma, and scirrhous cancer than in carcinoma simplex or medullare. Secondary changes such as fatty infiltration, leucocyte infiltration, hemorrhage, œdema, necrosis, etc., are of common occurrence in the interstitial connective tissue.

8. *Vascular Changes.* Obliterative endarteritis and periarteritis are of very frequent occurrence, and in the majority of instances are due to local changes. Proliferation of the endothelium of the lymph-channels was seen in a few cases. The vascular changes are most pronounced in the larger vessels.

9. *Changes in Nerve Elements.* Sections stained according to Weigert's method showed no change. Fujinami describes a connective-tissue increase in the nerve-trunks, but this was not seen in my material.

Special search was made for possible changes in the muscle-spindles, as these organs have not hitherto been considered in this connection. Forster studied the behavior of the spindle in many pathological conditions, such as acute myelitis, progressive muscular atrophy, etc. With the most marked change in the muscle he found the spindle remaining normal. Sherrington found no changes in the spindle to be produced by cutting muscles and tendons. Batten found a spindle which stained poorly in a case of tabes dorsalis, and in a case of atrophy and degeneration of the upper arm muscles, due to injury of the brachial plexus, the spindles were atrophic, the intrafusal fibres showed granular degeneration, the spindle sheath hazy, and the nerve-fibres of the spindle degenerated. Grünbaum found a peculiar hyalin deposit around the intrafusal fibres in a case of pseudohypertrophy. In my own work many preparations were examined for the study of the spindle. The nerve-trunks leading to the spindle were normal in every case examined. In one case of pectoral carcinoma longitudinal sections of a spindle showed a marked nuclear proliferation in the intrafusal fibres near the equator, but as similar appearances have been seen by several observers in apparently normal spindles the significance of this appearance is not clear.

10. *Small-celled Infiltration.* Leucocyte infiltration is found to some extent in or about all malignant growths. It is very marked in the case of epithelioma of the lip, but occurs to a very slight degree about invading cancer of the pectoral. The leucocytes are for the greater part collected about the bloodvessels and along the border-line of the invading growth. They are usually found intermingled with young tumor-cells, and it is difficult and often impossible to distinguish between the two.

11. *Occurrence of Mast-cells.* Both leucocyte and connective tissue mast-cells were at times found abundantly in the areas where small-celled infiltration was most marked. In a few instances mast-cells were found inside the sarcolemmar tube in company with other forms of leucocytes. In some of the cases of mammary cancer large numbers of eosinophiles were present in the areas of small-celled infiltration of the perimysium.

12. *Regeneration of Muscle.* In two-thirds of the cases of sarcoma and in three cases of epithelioma cell-forms identical with those seen in the repair of muscle were found in large numbers. They were never seen in the pectoral muscle in carcinoma, but were very numerous in a case of spindle-cell sarcoma of that muscle. The new fibres are found in the stroma of sarcoma and in the granulation tissue at the periphery of lip-epithelioma. The newly-formed fibres are usually short, and are spindle or club-shaped, or irregular in form. Their protoplasm stains well with eosin. The nuclei are numerous and are usually

grouped at one extremity. Many of the new fibres are vacuolated, their nuclei large and vesicular, and their protoplasm taking both nuclear and diffuse stains. These evidences of degeneration are exactly similar to those occurring in the new fibres after repair of muscle wounds. Fujinami, however, interprets the forms found near malignant tumors as being essentially degenerative in nature and not regeneration forms. He explained their formation as follows: The degenerating fibre is constricted at regular intervals, and this constriction goes on until the fibre is broken up into fragments, the nuclei at the same time proliferating ("ampullar atrophy"). The muscle-fragments he calls "ampullæ," and these he considers to be larger and to possess larger and more deeply-staining nuclei than the true muscle-buds in regeneration. While it is very probable that muscle giant-cells may be formed by the disintegration of the fibre, this explanation will not hold for my cases. In these, serial sections show that the giant-cells are really muscle-buds and not muscle-fragments. Their arrangement is not at all in accord with the idea that they are degenerating remains of old muscle. The presence of vacuoles in protoplasm and nuclei is also no argument that they are essentially degeneration forms, as it is well known that the newly-formed fibres in the repair of muscle wounds also quickly undergo degeneration, and do not lead to the production of muscle capable of functionating. I regard the appearances found in my cases as identical with those found in muscle repair.

Summary of Changes with Reference to the Difference Between Those Produced by Sarcoma and Carcinoma. 1. *Atrophy.* This is more marked in the case of carcinoma. In sarcoma the muscle may show an extreme degree of pressure atrophy at the border of the tumor, but in carcinoma there is usually a general atrophy, even of fibres at some distance from the cancer. This can be interpreted as chemical or toxic atrophy. I have not seen it in sarcoma.

2. *Hypertrophy.* This was not seen in any of the cases of carcinoma, but in a case of spindle-cell sarcoma of the pectoral the muscle-fibres appeared unusually large.

3. *Morphological Changes.* These are much more marked in the case of carcinoma, the muscle-fibres often assuming very grotesque forms. In sarcoma this distortion occurs to a very slight degree. The changes in carcinoma are so constant, even with the most careful technique, that they cannot be regarded as artefacts. The finding of mast-cells and leucocytes within the sarcolemma is also evidence that these changes occurred during life.

4. *Change in Striation.* Longitudinal fibrillation was present in all of the carcinoma cases; in sarcoma it was seen only rarely.

5. *Vacuolization.* This is a frequent and early change in both sarcoma and carcinoma.

6. *Retrograde Changes.* Early stages of cloudy swelling are common in the case of sarcoma, but in carcinoma the degeneration is more frequent and much more severe. Zenker's necrosis is of much more frequent occurrence in the case of carcinoma, in the pectoral often occurring at a distance from the tumor.

7. *Lacunar Erosion.* This form of degeneration of the muscle-substance is almost characteristic of carcinoma, being found only in one form of sarcoma, small round-cell of the skin. It may be emphasized once again that this so-called erosion is independent of any mechanical action of tumor-cells; but is a primary change in the muscle-substance.

8. *Nuclear Proliferation.* An increase in the number of the muscle nuclei is very commonly found in carcinoma, but often absent in sarcoma, even when other degenerative processes are present. This is in accord with the fact that the muscle-substance in sarcoma shows a much less tendency to retrograde changes. Nuclear proliferation goes hand-in-hand with degeneration of the muscle-substance. This apparent paradox may be explained on the hypothesis of a reversion to an embryonal state antecedent to degeneration, or that the normal muscle-substance has an inhibitory influence over the nuclei, the removal of which in degeneration of the former permits an unrestrained proliferation of the latter. Changes in size and form of the nucleus go hand-in-hand with nuclear proliferation.

9. *Sarcolemma.* Changes in this are more manifest in carcinoma because of the more marked changes in the protoplasm.

10. *Vascular Changes.* These are very common in both carcinoma and sarcoma; if anything, more marked in the case of the former. Hypertrophy of the endothelium occurs more commonly in sarcoma.

11. *Small-celled Infiltration.* This is very marked in lip-epithelioma, but in pectoral carcinoma it is very often very slight or entirely absent. In sarcoma the leucocytes were not numerous in the muscle and were usually limited to the perivascular areas of the perimysium. The enormous infiltration in lip-epithelioma may be due to secondary inflammation or infection, the location favoring the occurrence of these events.

12. *Muscle Regeneration.* As mentioned above, this was found only in the cases of sarcoma and lip-epithelioma. It was never seen in pectoral carcinoma.

From this summary it is evident that the muscle changes occurring near carcinoma and sarcoma are not identical, as claimed by Schaeffer and Fujinami. This observation is of great interest because of the significance which may be attached to it. The essential difference between the two may be briefly stated as follows: Retrograde changes are marked in the case of carcinoma; in sarcoma they occur to a very slight degree, or may be entirely absent. In sarcoma the atrophy is apparently only mechanical, while in carcinoma it occurs at a distance

from the tumor under conditions which make it very probable that it is of a toxic nature or due to a withdrawal of nutrition. The morphological changes in the muscle-fibre are more marked in carcinoma than in sarcoma, and this may be explained in the same way. Nuclear proliferation is marked in the case of carcinoma, in sarcoma it occurs to a slight degree, and in the majority of cases is absent. Regeneration forms are numerous in sarcoma, but were never found in pectoral carcinoma. Their presence in lip-epithelioma may be due to the extensive inflammatory changes present.

Comparing the changes produced in muscle by malignant tumors with those due to other causes, we find that the differences are of degree rather than of kind. Krauss, in experimental work on animals, found that sections of the nerve-trunks produced atrophy, nuclear proliferation, cloudy swelling, loss of striation, and vacuolization. Normal and diseased muscle-fibres were found side by side. Changes similar to these occur in acute anterior poliomyelitis, the bulk of the muscle being replaced by fat and connective tissue. In primary myopathy the fibres are atrophic or completely degenerated, occasionally hypertrophic (?) fibres are seen. The cross striation is indistinct, longitudinal fibrillation is marked, and fissuring, vacuolization, increase of connective tissue, fatty infiltration, and leucocyte infiltration are also present. In progressive muscular atrophy the fibres are much reduced in thickness, their striation is coarse or indistinct, with a tendency to fissuring; longitudinal fibrillation is common, and there is marked fatty degeneration, the sarcolemma often being filled with fat-droplets.

If the changes produced by malignant tumors show nothing specific in character, to what may the great difference in degree between those due to carcinoma and sarcoma be referred? The local changes in muscle invaded by tumors may be explained as due either to direct pressure of the tumor-cells or to chemical and nutritive influences. The difference between the two types may be the result of their different biological relations. Carcinomata are epithelial in origin, sarcomata mesoblastic, the invaded tissue muscle is also mesoblastic. An abnormal proliferation of any tissue means marked metabolic changes, the consumption of much nutritive material, and the giving off of much waste product. Tumor-cells behave in a manner similar to that of parasites, invading another complexus of cells, and flourishing at the expense of the latter. The tissue invaded suffers both from loss of nutrition and from the effects of the metabolic products of the more actively proliferating tumor-cells. Whether specific poisons are produced by the latter we are not yet in a position to say, but the changes about carcinoma are for the greater part to be interpreted as toxic in nature. In the invasion of muscle by sarcoma the antagonism between the two tissues must be less marked than in the case of carcinoma. The

sarcoma cells are genetically mesoblastic, and have similar capacities and tendencies to those of muscle. Hence, a sarcoma may infiltrate the entire perimysium and produce no changes except the simple atrophy due to mechanical pressure. In the central portion of a large sarcoma mass remains of muscle-fibres may still be present and may present no retrograde changes. Such an occurrence I have never seen in carcinoma; here the invaded muscle is quickly destroyed. The attempts at regeneration found in sarcoma are also an evidence of the less injurious effects of the latter. Carcinoma is, therefore, more antagonistic of muscle than sarcoma, because of its different biological relations, the different nutritive and metabolic conditions, and also probably because of the formation of poisonous products by the cancer-cells.

CONCLUSION. 1. The changes produced in striped muscle by invading malignant tumors are:

A. Retrograde.

1. Atrophy.
2. Morphological changes.
3. Changes in protoplasm.
   - Vacuolization.
   - Fissuring.
   - Longitudinal fibrillation.
   - Lacunar erosion.
   - Cloudy swelling.
   - Simple necrosis.
   - Zenker's necrosis.
   - Polychromatophilia.
4. Changes in nucleus.
   - Morphological changes.
   - Proliferation.
   - Vacuolization.
5. Perimysium.
   - Atrophy.
   - Hyperplasia.
   - Leucocyte infiltration.
   - Hemorrhage.
   - Œdema.
   - Necrosis.
6. Vascular changes.
   - Endarteritis.
   - Periarteritis.
   - Hypertrophy and hyperplasia of endothelium.

B. Regenerative.

1. Hypertrophy (?).
2. Regeneration forms.

2. These changes are not specific, but are produced by conditions other than malignant tumors.

3. The changes produced in muscle by invading carcinoma are more marked than those produced by sarcoma, but do not differ essentially in kind. Those produced by sarcoma are chiefly mechanical; those due to carcinoma are mechanical, nutritive, and metabolic. Evidences of toxic action are more marked in the latter than in sarcoma. Carcinoma acts more as an active injurious foreign body upon muscle than does sarcoma.

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A CASE OF OPHTHALMIA NEONATORUM CAUSED BY THE DIPLOBACILLUS OF MORAX AND AXENFELD.

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THAT the gonococcus is not the only cause of ophthalmia neonatorum is a conceded fact. The diplococcus, the streptococcus, the bacillus coli communis, and the micrococcus aureus also produce purulent conjunctivitis in the newborn. The last statistics of Groenouw show that in one hundred cases of ophthalmia neonatorum the gonococcus was present in only forty-one. All observers agree, however, in the opinion that when the disease is due to the gonococcus it usually assumes a very severe character.

Among the several micro-organisms described in connection with purulent conjunctivitis of the newborn the bacillus of Weeks and the diplobacillus of Morax and Axenfeld have never been mentioned. Having met with an unusual case of ophthalmia neonatorum caused by the last-named bacterium, which as a general rule is only found in cases of subacute catarrhal conjunctivitis, I have deemed it of sufficient interest to report.

Clinical History. E. L., female, three days old, child of healthy parents, presented slight œdema of the lids and a small amount of purulent exudate in both eyes, the symptoms being more marked in the right eye. There was no chemosis. The parents asserted that their two other children had shown, shortly after birth, similar symp-

toms, which, in the case of the elder one, were so severe as to threaten the destruction of one eye.

I prescribed protargol in 10 per cent. solution, increasing to 20 per cent. solution, and frequent washings with warm boric-acid solution, with the result that the œdema of the lids rapidly disappeared and the purulent exudate decreased very noticeably. I instructed the mother to continue the protargol instillations, and did not see the child during the following ten days. When called again I found that there was yet some purulent exudate in both eyes, and that the stump of the cord and the left ear were also suppurating. Otherwise the child seemed in perfect health. A much-desired bacteriological examination could not be made at the time. The suppuration of the ear and stump ceased under appropriate treatment, but the ophthalmia was rebellious. Noticing that not even applications of nitrate of silver in 2 per cent. solution had any effect, I made, at the first opportunity, a bacteriological examination of the conjunctival exudate, and found to my surprise a pure culture of the diplobacillus of Morax and Axenfeld. Knowing the almost specific action of zinc salts on this micro-organism, I prescribed sulphate of zinc in a 2.5 per cent. solution, to be instilled once a day. Under this treatment the ophthalmia, which had resisted all medication, got rapidly well. The right eye was cured in eight days, the left one in twenty. The disease had lasted in all nearly three months.

The history of this case will show, I hope, the importance of making an early bacteriological examination in all cases of ophthalmia neonatorum, and demonstrates also the fact that the diplobacillus described by Morax and Axenfeld as the cause of subacute catarrhal conjunctivitis can produce other forms of infectious ophthalmia.

FORWARD DISLOCATION OF THE HEAD OF THE FIBULA.

By J. F. Rinehart, M.D.,
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Forward dislocation of the head of the fibula is very rarely met with, and for very simple reasons. First, it is firmly bound in its articulation with the tibia by strong ligaments; and, second, there are no very powerful muscles attached to it anteriorly to either in whole or in part pull it from its place

Hamilton reports four cases observed by Savournin, Jobard, Thompson, and Dr. J. Hawley; while the *American Text-book of Surgery*, edited by Drs. Keen and White, says there have been seven cases of this injury reported.

It seems that while seven cases of forward dislocation have been reported at various times, there have been only five cases reported of backward dislocation of the same joint. This would appear at first thought to be the more common of the two, seeing the biceps femoris

is attached to the head of the fibula, and is a very powerful muscle. To my mind, it is extremely improbable that any case of dislocation of the fibula has ever occurred through muscular action alone in persons in whom the ligaments of the knee were in normal condition. If muscular action played a very important part in its production the backward dislocation would be much the more common of the two, while in reality it is not so often reported.

The treatment laid down by Hamilton for cases of forward dislocation is to flex the leg upon the thigh and the foot upon the leg at the same time pressure is made upon the dislocated bone. He says Thompson and Jobard were unable to effect reduction when the leg was extended.

In a case of my own I found the following:

The cause was direct violence, the limb having been caught between two freight cars in such a way that the tibia was pressed backward and the fibula forward. The symptoms were absence of the bone in its proper place and undue prominence in front of where the articulation should have been. The X-ray showed that the bone had been completely torn from its articulation and pulled forward. It was impossible to effect the slightest change in the position of the bones by traction upon the foot either alone or accompanied by pressure upon the head of the fibula. Reduction was accomplished by flexing the leg upon the thigh, the foot upon the leg, both as completely as possible, by making firm pressure upon the dislocated bone and by having extension gradually made while this pressure was increased. The bone slipped into place with considerable noise as soon as extension of the leg was begun.

A NOTE ON OSTEOPHYTES OF THE NASAL CHAMBERS.

By Alexander W. MacCoy, M.D.,
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It is some years since the subject of osteophytes in the nasal chambers first began to interest me. During the progress of an operation for deflection of the nasal septum, while I was making explorations in the nasal chambers, I became aware of certain sharp, rough, coral-like deposits situated within the nasal passages. As they proved to be neither necrotic nor rhinolithic aggregations, nor exostoses, I considered them osteophytes. These osseous bodies found in the nasal chambers have the anatomical arrangement and composition of osteophytes. Their formation being arranged in laminated or radiated structures, such anatomical arrangement determines the fitness of the name.

I have neglected to investigate the literature on this subject, having

found only one plate where such bodies in the nasal chambers were illustrated.[1]

To enable one to determine definitely the existence and location of osteophytes, a *digital* exploration of the nasal chambers is necessary. Some of us can remember a paper by Dr. Harrison Allen in which he advocated *digital* manipulation of the anterior regions of the middle turbinated bodies for certain diseased conditions present. Following this suggestion, I have for some years used the little finger as an intelligent exploratory probe. In all my operations for deflection of the nasal septum I have found it necessary in such cases to use the finger far back in the nasal cavities to enable me to get a clear idea of the trend of the septum, and also of its thickness. While exploring in this manner I have found in many instances these bony concretions, which I have named osteophytes.

The clinical fact is interesting that bony deposits do in a certain number of cases exist in the nasal chambers without definite anatomical relation to the normal osseous formations.

The presence of osteophytes in the nasal cavities materially interferes with the success of an operation for nasal deflection by preventing free respiration and drainage, and, especially when they are large, by hindering complete manipulation of the septum after the incisions have been completed.

Osteophytes generally are to be found well back in the nasal chambers, situated underneath the lower turbinated bone, on the bony septum, and on the floor of the nose. In size they vary from a sharp spicula to a small, irregular mass. While formulating these notes a young woman came under my care who was a subject of osteophytes in various parts of her body. Large masses were present on the tibia, as well as on the lower end of the radius. The subject consulted me for deflected septum. An operation had been performed upon her some time before for deflected septum, but it had been unsuccessful. Examination showed a marked deflection of the triangular cartilage, associated with deformity of the lateral cartilages. In the triangular cartilage plaques of bony tissue could easily be felt, which were undoubtedly osteophytes. After the second operation there was troublesome bleeding which was not controlled by the tubes, but required tamponing. This incident was, evidently, the result of the interstitial change over the cartilage involving the coats of the bloodvessels and preventing their contraction. The results of the second operation were most satisfactory, and, after a lapse of over a year, the alignment of the septal wall is perfect. In several other cases of osteophytes present in cases of deflection of the nasal septum coming under observation the bony concretions were

[1] Zuckerkandl, "Anatomie der Nasenhöhle," Table I., Fig. 2, Description, p. 187.

well back in the nasal passages, and were broken off and removed during the operation for deflection. I recall one case in a young woman where the osteophytic deposit was near the posterior naris, where it could barely be reached by the tip of the little finger. If this bony mass had not been discovered, complete restoration of the respiratory function of that side would not have resulted, as was the case after the osteophytes had been removed and the deflection corrected.

SUMMARY. 1. Under an anæsthetic, with the little finger as an intelligent probe, certain osseous structures foreign to normal nasal conditions may be found.

2. That these osseous masses are osteophytes, having the anatomical structure of such bodies.

3. That osteophytes, clinically, are *new* conditions to be considered and studied.

4. That in operations for deflection of the nasal septum, lack of complete success may result from their presence.

5. That osteophytes are loose in structure and readily removed by proper instruments.

A REVIEW OF SOME RECENT LITERATURE ON CERTAIN INFECTIVE DISEASES.

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SYPHILIS. The occurrence of fever as a manifestation of syphilis has been known for some time, it having been commented upon especially by Musser and by Osler. More recently, Futcher,[1] writing on the subject, states that in all cases of fever of obscure origin the possibility of the fever being syphilitic in nature should be borne in mind, and that careful examination of the long bones, skin, and viscera should be made, with a view to detecting manifestations of syphilitic infection. He states, also, that the fever of syphilis may occur at different periods during the course of the disease: 1. It may occur in very rare instances three or four weeks before the onset of the secondary skin eruption. This fever is very puzzling and, until the appearance of the eruption, it is likely to be attributed to some other cause. 2. It may precede or be coincident with the appearance of the secondary eruption. This is the so-called "fever of invasion," and it is a very common and important symptom of secondary syphilis. 3. The fever

[1] Syphilitic Fever. New York Medical Journal, 1901, vol. lxxiii. p. 1065.

may occur at any time during the course of the secondary or tertiary stages. The late occurrence of the fever is the most interesting feature. In one of the cases reported it occurred twenty-nine years after the disease had been contracted. It is said that the fever of invasion is rarely absent at the onset of the secondary symptoms. It usually precedes the appearance of the secondary eruption by a week or ten days; rarely by more than two weeks. It is generally believed to be a symptom of the invasion of the system by the organism the cause of syphilis or by its toxic products. The degree of fever varies in different cases; it may be moderate (101° F.), but sometimes it is much higher (104° to 105° F.) in the afternoon. The fever of invasion, as well as the syphilitic fever associated with the late manifestations of the disease, may present any one of the three following clinical types: (1) A mild continuous pyrexia, in which the temperature ranges in the neighborhood of 101° F.; Osler states that this type is not uncommon in the fever that ushers in the constitutional symptoms; (2) a remittent type of fever, with morning drops toward normal and evening exacerbations —the usual fever of invasion; (3) a definite intermittent fever; this is the most remarkable form of all, and it is the type that is most likely to lead to diagnostic errors. It is sometimes accompanied by chills and fever. The febrile diseases for which syphilis is often mistaken are the malarial fevers, typhoid fever, tuberculosis, and sepsis. When general pains and joint pains accompany the fever the diagnosis of rheumatism may be made. In doubtful cases the true nature of the fever may be demonstrated readily by instituting anti-syphilitic treatment.

Carriere,[1] writing of syphilitic fever, states that it is extremely rare (a statement that will not meet concurrence on the part of those who see much of the disorder), and that during the past ten years he has observed but two instances of it. The fever is said to develop usually within forty to sixty days after the initial sore, but sometimes much later. Women especially are likely to have the fever, probably, it is believed, because the initial lesion may go undetected and untreated, and a severe infection thus occur. The febrile affections for which the syphilitic fever may be mistaken are malaria, psittacosis, cerebro-spinal meningitis, miliary fever, pyemia, verrucose endocarditis, miliary tuberculosis, recurrent fever, and influenza. It is only in the most unusual cases that diagnostic difficulties are likely to arise, but the differentiation from typhoid fever may be perplexing. Attention to the following will facilitate the diagnosis: 1. A coated, moist tongue in an individual that presents a typhoid aspect suggests an abnormal typhoid fever or another disease. 2. Extraordinary intensity of the headache suggests syphilis. 3. Very profuse perspiration bathing the body of the patient

[1] La typhose syphilitique. Gazette des Hôpitaux, 1901, vol. lxxiv. p. 65.

is unusual in typhoid fever. 4. Extensive and generalized eruption suggests that the disease, if it be typhoid fever, is a very unusual form. 5. The failure to detect the typhoid bacillus in the stools or in the fluid obtained by puncture of the spleen (to my mind an unjustifiable procedure) suggests the absence of typhoid fever. Examination of the blood is of great value; in syphilitic fever there occur greater oligocythæmia, microcythæmia, and oligochromæmia than in typhoid fever, and hyperleucocytosis occurs in syphilitic fever, and hypoleucocytosis in typhoid fever. 6. The absence of the Gruber-Widal reaction in all stages of the disease is of great value. Finally, one should seek for evidences of syphilis—the initial lesion or a cicatrix (sometimes difficult to find, especially in women), and evidences of syphilis in the viscera and the skin, etc. In case it be suspected that a syphilitic patient has acquired typhoid fever, the following are of diagnostic value: (1) The absence of the Gruber-Widal reaction; (2) the absence of the typhoid bacillus in the stools and the fluid obtained by puncture of the spleen; and (3) the therapeutic test—the result of anti-syphilitic treatment. It is believed that the syphilitic fever is an expression of syphilitic infection of an organism of lessened resistance.

Scheib[1] reports the case of a man, aged fifty-one years, who, three months before his death, suffered an injury in the region of his stomach. Soon thereafter he complained of pain, vomiting, loss of appetite, hæmatemesis, and the passing of blood by the bowel. Examination revealed the ordinary signs of bronchitis, and, in addition, pain, tenderness, and rigidity about and below the umbilicus—all of which were relieved by evacuation of the bowels. Subsequently fever, right-sided pleuritis, hæmoptysis, collapse, and death occurred. The necropsy revealed a half-litre of fresh blood between the stomach and the liver, and another half-litre between the stomach and the spleen; a funnel-shaped, perforating ulcer, the size of a dollar, on the anterior surface of the stomach, toward the hilum of the spleen; a dense infiltration, with central necrosis, about the size of a dollar, in the region of the pylorus; similar areas of infiltration at eight different regions of the small intestine—some perforated, but all closed by adhesions with the surrounding structures; and many areas of infiltration, grayish-white in color, of firm consistence, without central necrosis, varying in size up to that of a hen's egg, and situated in the lungs. The possible nature of the affection is discussed with especial reference to the likelihood of its being neoplastic or tuberculous, and it is pointed out that it cannot be the one or the other; that tubercle bacilli could not be detected; that microscopically the infiltration consisted of granulation tissue with giant cells; and that the bloodvessels revealed marked endar-

[1] Syphilis mit gumöser Affektion des Magens und Darms so wie der Lungen bei einem 51 Jährigen Manne. Prager medicinische Wochenschrift, 1900, vol. xxv., Nos. 45, 46.

teritis proliferans that in some places had progressed to complete closure of the vessels. From these facts it is concluded that the case is one of visceral syphilis.

Einhorn[1] divides syphilitic affections of the stomach into three groups—syphilitic ulceration, syphilitic tumor formations (gummas), and syphilitic stenosis of the pylorus. Illustrative cases of each group are cited. Of interest is the fact that two of the cases of obstruction of the pylorus, in consequence of the failure of the ordinary methods of treatment, would have gone on to operation had not the possibility of their syphilitic nature been borne in mind. It is pointed out that syphilis of the stomach is more common than is generally thought; that tertiary syphilis may give rise to severe disturbances of the gastro-intestinal tract; that in all severe disturbances of the gastro-intestinal tract the possibility of syphilis should be considered; and that such disturbances are amenable to antisyphilitic treatment. A review of cases reported in the literature is given.

Mracek[2] states that dactylitis is uncommon in acquired syphilis and in adult life, whereas, as a manifestation of hereditary syphilis in infancy it is quite common. Among 50,000 syphilitic patients he has observed five cases of dactylitis—two in adults and three in children. He reports in detail a case occurring in a man, aged thirty years. Careful investigation, including the use of the X-rays, convinced him that the disease develops primarily in the bone, which becomes rarefied, with the coincident formation of peculiar spaces filled with colloid infiltration and exudation masses; and that, secondarily, the joints and the softer tissues become implicated. The deformities are due principally to inflammatory and œdematous infiltration of the softer tissues. The affection is almost painless, chronic in its course, and tends to heal, with the production of deformities—ankylosis or flail joints. Treatment should consist in administering large doses of potassium iodide (75 grains daily) for a number of weeks. Mercury may be combined with the iodide, but care must be exercised in its use, as in syphilitic diseases of bones its employment is sometimes attended by unfavorable results.

Syphilis and Tuberculosis. Dieulafoy[3] reports two cases of concomitant syphilitic and tuberculous disease of the lung. He states that when a subject for a long time syphilitic (in one of his cases twenty years) becomes affected with tuberculosis of the lungs the prognosis is good, and especially is the tuberculosis likely to pursue a favorable course. He warns against the use of potassium iodide and urges the use of mercury. He suggests the alternate use, for periods of fourteen days, of

[1] Ueber Syphilis des Magens. Archiv für Verdauungskrankheiten, 1900, vol. vi. p. 150.
[2] Zur Dactylitis syphilitica. Wiener klinische Wochenschrift, 1900, vol. xiv. p. 432.
[3] Association de la tuberculose et de l'syphilis. La tribune médical, 1901, No. 13.

daily injections of biniodide of mercury (0.004 to 0.008 gramme) and of daily injections of cacodylate of sodium (0.05 gramme).

TUBERCULOSIS. At the final session of the recent British Congress on Tuberculosis the following resolutions were unanimously adopted: 1. That tuberculous sputum is the main agent for the conveyance of the virus of tuberculosis from man to man. Indiscriminate spitting, therefore, should be suppressed. 2. All hospitals and dispensaries should present every out-patient with a leaflet on the prevention of consumption, and insist on the use of a pocket spittoon. 3. Notification of tuberculosis should be established when possible. If compulsory notification is impracticable, voluntary notification should be encouraged. 4. The provision of sanatoria is an indispensable part of measures for the diminution of tuberculosis. 5. Medical officers of health should use all their powers and relax no effort to prevent the spread of tuberculosis by milk and meat. 6. In view of the doubt thrown on the identity of human and bovine tuberculosis, the government should be requested to institute an inquiry into the subject. 7. The educational efforts of the great national societies for the prevention of tuberculosis are deserving of support. 8. A permanent international committee should be appointed to report on the measures for the prevention of tuberculosis in different countries, to publish a popular statement of these measures, and to keep and publish a record of scientific research in relation to the disease. 9. Overcrowding, defective ventilation, and the damp and insanitary dwellings of the working classes diminish the chances of curing consumption and are predisposing causes of the disease. 10. The attention of governments and charitable persons should be called to the necessity for establishing anti-tuberculosis dispensaries.

Friedmann[1] publishes the results of an extensive series of investigations concerning the significance of the tonsils as a portal of entry for tuberculosis in young children. The investigations comprise the examination of the tonsils obtained at 91 necropsies, and of tonsils removed by operation from 54 children, with one exception under five years of age. In cases 1 to 3 the tonsillar tuberculosis probably was not primary; in cases 4 to 7, and in case 9 the tuberculosis was most likely primary in the tonsils and secondary in the lymphatic glands, intestine, and bone; in case 8 the tonsils, which contained many tubercles, with giant cells and bacilli, were the only seat of tuberculosis in the body; in cases 10 to 16 (2 with and 5 without tuberculous lesions elsewhere in the body) the tonsils contained typical giant cells, but bacilli could not be demonstrated; in cases 17 to 19 the giant cells in the tonsils were attributed to some process other than tuberculosis; in cases 20 to 30 (cases of

[1] Ueber die Bedeutung der Gaumentonsillen von jungen Kindern als Eingangepforte für die Tuberculöse Infektion. Ziegler's Beiträge zur path. Anatomie, etc., 1900, vol. xxviii. p. 66.

extensive tuberculosis) the tonsils revealed no tubercles, but the presence of old cicatrices suggested the probability of previous tuberculosis; in cases 31 to 34 tuberculosis of the internal organs was unassociated with tuberculosis of the tonsils; in cases 35 to 37, although the tonsils presented no tuberculous alterations, tubercle bacilli were demonstrated in smear preparations made from the surface of the organs; in the remaining 54 cases tuberculosis was found nowhere in the body. In only one of the 54 cases of operative removal of the tonsils from living children were tuberculous lesions detected that could be looked upon as primary. It is believed that although strong children with chronic hypertrophy of the tonsils rarely have tuberculosis of the tonsils, tuberculosis of the tonsils in children in general is quite common; and that it is not likely that infection occurs by means of the lymph-stream, the blood-stream, or by inhalation, but rather by means of the food. Following the primary infection there occurs a descending tuberculosis of the lymph glands, the result of infection through the lymph-stream.

Influenza. Michel,[1] writing of the pulmonary complications of influenza, gives expression to some opinions that evidently are founded more on speculation than on morbid anatomy. In the pathogenesis of these complications especial significance is attached to atmospheric influences as well as to predisposition, such as infections and intoxications, more especially alcoholism. Distinction is drawn between primary complications (due to the influenza bacillus) and secondary complications (due to mixed infection). The primary complications comprise the simple catarrh, the acute bronchitis, "fluxion de poitrine," a congestion simulating diaphragmatic pleuritis, and a "hæmoptytic congestion;" the secondary complications comprise splenopneumonia, bronchopneumonia, and pneumonia. To many of us such terms are unusual, at the least. According to Michel, the "fluxion de poitrine" is of sudden onset, and implicates the lungs, the pleura, chest wall, and the nerves. The patient complains of marked pain, aggravated by deep inspiration, coughing, and pressure, and mucous, mucopurulent, and sometimes sanguinolent expectoration. Examination reveals dulness, diminished or rough vesicular breathing, and friction-sounds. Pleural exudation may occur, and the congestion may progress to hepatization of the lung; the process, however, usually is confined to congestion. This "fluxion de poitrine" is not uncommon in French literature, and is said to be a process midway between "congestive bronchitis" and bronchopneumonia. In America, at least, such condition is not recognized. The ordinary "fluxion de poitrine" we should recognize probably as a bronchopneumonia, and should the condition progress to hepatization we should probably interpret the phenomena

[1] Des complications pulmonaires de la Grippe. Le Bulletin Médical, 1901, vol. xv. p. 285.

as indicating the confluence of bronchopneumonic foci. Nor can it be said that a form of congestion of the lungs simulating diaphragmatic pleuritis is recognized in America. Michel recognizes it by the occurrence of pains at the lower portion of the thorax (the insertion of the diaphragm), pain in the neck (from implication of the phrenic nerve), hiccough, sometimes uncontrollable vomiting, and more or less evidences of inflammation of the lower portions of the lungs and pleuræ. His "hæmoptytic congestion" is of interest in that in case of severe hemorrhage the condition may be mistaken for pulmonary tuberculosis. It is difficult to understand what is meant by "splenopneumonia." Attention is directed to certain peculiarities of the course of influenza pneumonia, especially to the insidious onset and to the difficulty sometimes experienced in distinguishing it from typhoid fever, especially pneumotyphoid. The Gruber-Widal reaction is said to be of value, and to this might have been added the examination of the expectoration for the influenza bacillus. Mention is made of the occurrence of delayed resolution, of abscess of the lung, of gangrene of the lung, and of a condition designated "bronchoplegie á signes pseudocavitaires." This condition, which we should probably designate bronchiectasia, is termed bronchoplegia because it is thought that the stagnation of the bronchial secretion and the excessive suppuration in the dilated bronchi are due to paralysis of the bronchial muscles. The condition is of importance in that it may be mistaken for abscess or tuberculous cavity formation in the lung, both of which conditions have a much more serious outlook than has the "bronchoplegia."

Castellani[1] has examined bacteriologically nine cases of complications and sequels of influenza—pneumonia, bronchopneumonia, pleuritis, empyema, suppuration of the ear, meningitis, arthritis, and enteritis. The investigations comprised bacteriological examination of the expectoration, pleural exudate, joint fluid, cerebro-spinal fluid, lung "juice," and the blood. Influenza bacilli were found only in the expectoration and in the pus from the ears, and not in pure culture, but mixed with pneumococci in excessive numbers. Animals inoculated with these fluids died of pneumococcic sepsis. Examination of the cerebro-spinal fluid led to indefinite results. The remaining secretions and the blood revealed only pneumococci. In addition, Castellani examined the blood in twenty-one cases of typical influenza, and in a number of other cases with complications. In all cases of pneumonia and bronchopneumonia he found only pneumococci. It is believed that the influenza bacillus may be the cause of the complications of influenza, but that, as a rule, this bacillus does not enter the circulation, and that many complications are produced by the pneumococcus.

[1] Contributo allo studio bacteriologico di alcune complicanze dell' Influenza. Riv. crit. di clin. med., 1901, No. 3. Fortschritte der Medicin, 1901, vol. xix. p. 781.

In contrast with these opinions, the opinions of Jehle[1] may be cited. This writer details the results of his investigation of a number of cases of scarlet fever, measles, varicella, pertussis, diphtheria, and a number of different diseases in adults, with reference to mixed infection with the influenza bacillus. It was ascertained that influenza is a common secondary infection in childhood, and that it may occur early in the course of the primary disease. The influenza infection usually is in the lower respiratory tract, but it may remain localized to the tonsils, and it may assume an epidemic character. With the occurrence of influenza mixed infection an increased morbidity and mortality develops. In the acute exanthemas there occurs regularly an invasion of the blood-stream with the influenza bacillus, which may enter from the tonsils (this statement is of especial importance and interest as contrasted with the observations of Castellani, previously cited). This bacteræmia may occur very rapidly, and in scarlatina it may be observed even before the eruption. In the non-exanthematic diseases and in adults the influenza bacillus is but rarely found in the blood. If, however, influenza bacteræmia does occur, severe organic lesions—carditis, endo-pericarditis, pleuritis, brain abscess, etc.—usually occur also. It is believed that the exanthemas in some manner predispose the body to infection with the influenza bacillus and facilitate the entrance of the influenza bacillus into the blood-stream.

Franke[2] states that he has observed with great regularity in influenza a striated redness of the anterior faucial arch, whereas the uvula, the lateral part and about two-thirds or three-fourths of the velum palati, and sometimes the free edge, retain their normal color. This is said to be especially valuable as a diagnostic sign in cases of chronic influenza. He has observed also swelling of the anterior papillæ of the tongue—a swelling exceeded in degree only in cases of scarlatina and in a few cases of measles. This swelling may occur on the second or third day of the disease, and possesses the same diagnostic significance as does the redness of the faucial arch. Finally, attention is directed to the diagnostic value of enlargement of the spleen, said to occur especially in the chronic forms of the disease.

Federn[3] states that in influenza the blood pressure (measured with von Basch's sphygmomanometer) is abnormally low, that this results from disturbance of the cardiac nerves, and that it occurs so constantly that it is of diagnostic value. Inferentially, it is said that most of the symptoms in influenza are due to defective circulation—that they are quasi congestive symptoms. The blood pressure is thought to have

[1] Zeitschrift für Heilkunde, 1901, vol. xxii. p. 190.

[2] Ueber ein typisches Influenzasymptom, die Influenzaangina, und über Influenzazunge und Influenzamilz. Deutsches Archiv für klinisches Medicin, 1901, vol. lxx. p. 280

[3] Ueber Influenza. Wiener medicinsiche Wochenschrift, 1901, vol. i. p. 1162.

prognostic value in influenza, in that those patients that have high pressure before the attack (in consequence of increased resistance of the vessel wall) are less resistant and recover from the disease slower than do others.

Petrucci[1] reports a number of cases of influenza during the course of which there occurred hemorrhages of different sorts—epistaxis, hæmoptysis (of all grades from slight tinging of the expectoration to profuse hæmoptysis), metrorrhagia, hæmaturia, etc. He believes that one is justified in speaking of a hemorrhagic form of influenza in addition to the catarrhal, the nervous, and the gastro-intestinal forms, and he finds justification for this not only in the occurrence of the hemorrhages, but also in the fact that we already speak of hemorrhagic typhoid fever, hemorrhagic scarlatina, etc., and in the fact that the development of hemorrhage in influenza is indicative of a malignant type of the disease and influences materially the prognosis. In these cases convalescence is much prolonged if the hemorrhage does not lead directly to the death of the patient, as occurred in one of the cases reported.

Huber[2] states that recently following an epidemic of influenza in which all of the seven members of a family were affected, four suffered with suppurating buboes and lymphangitis. Of these, three (not the father) previously had been vaccinated, and the buboes were confined to the glands of the axilla. It is presumed that these glands were a locus minoris resistentiæ from the vaccination. The father's bubo was in the inguinal region. The buboes contained only streptococci—no influenza bacilli or plague bacilli. Recovery ensued in all cases. It is presumed that, aside from rendering the glands less resistant, the vaccination had nothing to do with the infection—a hæmatogenous general infection with the streptococcus. This opinion is borne out by the situation of the bubo in the father. The case is reported as an instance of heterochronous polyinfection—that is, mixed infection with the influenza bacillus and the streptococcus.

Ford[3] recently has written of acute aortitis occurring in the course of influenza, of which condition he has observed eighteen cases. In its manifestations it is said to resemble the condition as it occurs in other acute infectious diseases, notably typhoid fever. It affects especially patients past thirty years—those, therefore, whose arteries perhaps already show some of the consequences of wear and tear. It involves especially the arteries of the legs; usually those of one leg, but sometimes those of both. It is most likely to develop during convalescence from influenza, but it has been noticed to occur some time after apparent recovery. The onset usually is sudden and manifested by pain (that

[1] Ein Beitrag zur Casuistik der Hemorrhagien bei Influenza. Wiener medicinische Presse, 1900, vol. xli. p. 1770.

[2] Ueber eine Familiäre Streptococcenerkrankung nach Influenza. Correspondenzblatt für Schweitzer Aerzte, 1901, vol. xxx. p. 233.

[3] Thèse de Paris, 1901.

may be so severe as to require morphine), tenderness along the course of the inflamed artery, paræsthesia (that may even precede the pain), lowering of the temperature of the affected part, and mottling of the skin. Arterial obliteration is indicated by cessation of pulsation below the site of obstruction. This may be temporary and followed by restoration of the circulation without damage to the part; or it may be permanent and followed by gangrene. The gangrene usually is of the dry variety, but it may be moist if there be associated phlebitis. In the event of gangrene there may occur sphacelation of the gangrenous area and recovery, or there may occur general infection and death. Of fifteen patients, six died. As regards treatment, it is recommended to encase the affected limb in wadding and splints, and to apply methyl salicylate or an ointment of salicylic acid, turpentine, and belladonna. The pain, if severe, should be relieved by morphine, and in the event of certain complications surgical intervention is called for.

Diemer,[1] writing of influenza polyneuritis, states that usually it develops during convalescence (as does diphtheritic neuritis), within from ten days to one month from the beginning of the infection. The initial manifestations may be either sensory or motor; the latter occur constantly without any special predilection for particular muscles. While all the muscles may become affected, the muscles of the upper or the lower extremity of one side or of the trunk, or of the neck, are most frequently affected. Frequently, also, the muscles of the face and the muscles supplied by the vagus are affected. Less frequently the oculomotor, the abducens, the optic, and the auditory nerves are affected, and exceptionally the nerves of the palate, the pharynx, and the diaphragm. Paralyses that involve the extremities diminish in intensity as the trunk is approached. Reactions of degeneration are constant. Ataxia is common, and the gait may resemble that of a tabetic; flaccid paralysis is constant. Disturbances of sensation are more or less pronounced; they are usually less intense than are those that occur in other polyneuritides, notably the alcoholic. Trophic and vasomotor disturbances are not conspicuous. The skin and tendon reflexes are diminished or absent. The course of the affection may be acute or subacute. General and localized forms may be distinguished. Complete recovery usually occurs; in some cases, however, more or less atrophy of certain groups of muscles persists. The treatment, as a rule, should be symptomatic; but especial attention should be directed to hygienic and dietetic measures, that recurrences may not occur.

Pneumonia. Schultz[2] directs attention to the fact that during recent years increasing support has been given the view that croupous pneu-

[1] La polynévrite grippale. Gaz. hebdom. de méd. et de chir, 1901, vol. xlviii. p. 37.

[2] Contribution a l'etude de la pneumonie fibrineuse. Archives des sciences biologiques de St. Petersbourg, 1901, vol. viii. p. 1.

monia is due to a primary pneumococcic infection of the blood and a secondary infection of a lung that has become reduced in resistance rather than to a direct infection of the lung through the respiratory passages. In an attempt to confirm this view through experimental investigation he injected pneumococci into the jugular veins of lower animals, and after their death he examined their lungs microscopically, even when they presented no naked-eye deviations from the normal. He states that the lesions of pneumonia were evident in all the lungs, and that these lesions corresponded to those of red hepatization in the human subject. The bloodvessels of an entire lobe or of some of the lobules were dilated and contained many leucocytes (mostly mononuclear) and many pneumococci. The walls of the alveoli were infiltrated with young cells, and the alveolar epithelium had desquamated. In the lumen of the alveoli considerable quantities of fibrin formed early; in the later stages this was mixed with erythrocytes, so that in some instances the lumen of the alveoli was completely filled. It is concluded that pneumonia may be induced in rabbits merely by injecting pneumococci into the blood-stream—that is, without previous alterations in the lung. Regarding the question how infection of the blood occurs in man—that is, infection of the blood previously to infection of the lung—an opinion is withheld.

Typhoid Fever. Recently a number of studies of what may be termed the nervous manifestations of typhoid fever have been published. Thus Debove,[1] directing attention to the fact that most of the symptoms of typhoid fever are nervous in nature, and are to be attributed to the action of the typhoid toxin on the nervous system, reports the case of a man, aged twenty years, who, in September, 1900, passed through an attack of typhoid fever with intestinal hemorrhages. At the end of about two months the patient was considered well enough to be discharged from the hospital, but that same day he became ill with lumbar pain, fever, and delirium, said to have resulted from mental excitement. The lumbar pain increased in severity until about the middle of December, when after a short walk it became so severe that the patient was unable to move his lower extremities. With rest in bed the pain abated somewhat, but it recurred with severity as soon as any attempt at movement was made. Examination revealed motor, sensory, trophic, and vasomotor disturbances. The lumbar pain was much increased by pressure over the sacrum. Spontaneous pain or disturbances of sensation were not present in the lower extremities, but on attempting to walk there occurred severe pain extending from the lumbar region through the legs. The legs were distinctly paralyzed and atrophied. The temperature on the first day ranged between 38° C. and 38.5° C.,

[1] Sur un cas de paraplégie post-typhique. Le Bulletin Médical, 1901, vol. xv. p. 141.

on the seventh day it was 40.4° C., and on the twelfth day it reached normal. At this time the lumbar pains and the other manifestations gradually subsided. At the time of the report, when at rest the patient had no complaints; when he attempted to walk, however, slight pains in the lumbar region occurred. He was able, nevertheless, to ascend stairs without especial difficulty. Sensation was normal, but in the lower extremities there was distinct atrophy. The case is considered an instance of post-typhoid paralysis due to circumscribed inflammation of the lower portion of the spinal meninges.

Foulerton and Thomson[1] state that the nervous symptoms of typhoid fever may be due either to the direct action of the specific bacillus or to the action of secondary invaders. It is difficult to determine clinically the range of activity of each of these, though it is stated that in the beginning of the disease the typhoid bacillus alone is probably the active agent; later, other bacteria may play a rôle, as we know they are active in producing the intestinal lesions. The symptoms referable to the typhoid bacillus may be produced by either the bacillus acting directly or by its toxin. The toxæmia may give rise to manifestations of severe irritation of the brain, which sometimes can be distinguished from the meningitis due to the typhoid bacillus or other organisms only by the onset of paralysis or optic neuritis. Two cases illustrating the two forms of cerebral disturbance are reported. The first case was that of a girl, aged nine years, who suffered from severe toxæmia. She was noisy and restless, passed into a low, muttering delirium, and died. No alterations were found in the brain, and cultures from the brain gave negative results. The second case was that of a boy, aged twelve years, who was thought to be suffering from tuberculous meningitis until the Gruber-Widal reaction was obtained. The necropsy revealed marked congestion of the meninges, and bacteriological investigation revealed the typhoid bacillus. This is therefore a case of true typhoid meningitis, of which it is said that eighteen cases have been reported. The writers undertook the study of the action of the typhoid toxin on the ganglion cells of the central nervous system by injecting living cultures of the typhoid bacillus and the typhoid toxin into rabbits, and studying the brain lesion. Aside from occasional slight swelling and indistinctness of the details of the cells, no noteworthy alterations were detected; from which it is concluded that typhoid infection or typhoid toxæmia does not produce noteworthy discoverable alterations in the ganglion cells of the nervous system.

A year or more ago, at the German Hospital, I performed a necropsy on a subject dead of fibrinopurulent meningitis complicating typhoid fever. From the exudate the typhoid bacillus was obtained in large

[1] The Causation of Nervous Symptoms in Typhoid Fever. Lancet, 1900, vol. i. p. 1121.

numbers and in pure culture. The case will be reported shortly by the assistant pathologist to the hospital, Dr. Edward Kemp Moore.

Salomon[1] points out that he has observed that in a number of cases of typhoid fever the optic disk presents an appearance suggesting cerebral tumor—not a distinct papillitis, but a veiling of the disk, with indistinctness of its edges, injection of the capillaries, dilatation and tortuosity of the veins. Performing lumbar puncture, he found the spinal fluid under high pressure—from 180 to 250 mm. of water. The fluid was sterile, contained about the normal amount of albumin and of leucocytes, and did not cause agglutination of the typhoid bacillus, although in each of the three cases investigation of the blood-serum did cause such agglutination—in one case even when diluted 1 to 500. It is believed that an acute serous meningitis is very common in typhoid fever, and that to this is due one of the characteristic features of the disease—the slow pulse. Spinal puncture was followed by subsidence of the headache.

Stadelman[2] points out that in his previous communication on lumbar puncture he referred to the symptoms of brain pressure not only in typhoid fever, but also in pneumonia, scarlatina, etc., and he attributes them to an angioneurotic hydrocephalus the result of the action of the typhoid toxin. He has not observed the good effects of lumbar puncture reported by Salomon.

Kühn[3] reviews briefly the previously reported cases of typhoid spondylitis, and reports a personal observation of his own. The case was an instance of severe typhoid infection in which, thirty days after the temperature had become normal, vague pains developed in the lumbar region. Gradually a circumscribed swelling and marked tenderness on pressure developed in the region of the lumbar vertebræ, and these were associated with excessive pain on motion of the trunk. In the course of about a week a distinct lumbar kyphosis developed, but symptoms of compression of the spinal cord did not appear. Gradually the pain and tenderness lessened and the kyphosis disappeared, so that at the end of about three months the patient could be discharged from the hospital. It is stated that, according to Quincke, who first described typhoid spondylitis, its principal characteristics are tenderness of certain spinous processes and manifest swelling over the affected part. To these Kühn adds the kyphosis, which, however, has been observed in but few cases, and is to be attributed probably to destructive processes in the body of the vertebræ. Treatment should consist in absolute rest in bed and the local application of cold. In the event of serious symptoms antiphlogistic remedies may be called for—even in some cases orthopedic appliances.

[1] Berliner klin. Wochenschrift, 1900, No. 6.
[2] Ibid., 1900, No. 8.
[3] Ueber Spondylitis typhosa. Münchener medicinische Wochenschrift, 1901, vol. xlviii. p. 926.

Mannini[1] directs attention to what he believes to be the rather common occurrence of peritonitis in typhoid fever in the absence of perforation of the intestine. He states that in typhoid fever the intestinal epithelium has lost its protecting power; that the lymph vessels of the intestine are inflamed, and that this inflammation extends not only to the serous covering of the intestine, but even to the mesenteric lymph glands; that the typhoid bacillus is present in the spleen, the liver, and the kidney; and that as it finds its way to these organs so also does it find its way to the peritoneum. It is believed that the reason that peritonitis does not occur more frequently in consequence of such infection of the peritoneum by the typhoid bacillus is because the peritoneum possesses marked absorptive powers and is able to destroy bacteria such as the staphylococci, the bacterium coli commune, and the typhoid bacillus. This bactericidal property is associated with the presence of leucocytes and the peritoneal serum. It is believed that there occurs in the course of typhoid fever and during convalescence an acute peritonitis that is not a perforative peritonitis; that this is due to propagation and diffusion through the intestinal wall of the specific infective agent; that such propagation and diffusion occur in every case of typhoid fever, but that peritonitis results only when the resorptive and bactericidal properties of the peritoneum have been overcome. Such peritonitis is said to give rise to symptoms similar to those of perforating peritonitis, from which it is to be distinguished by the absence of gas in the peritoneum. It gives a better prognosis, and its treatment is medicinal.

Osler[2] has drawn up the following schedule of specific directions to be followed in cases of typhoid fever in which perforation is suspected; though intended especially for use in hospital practice, they are of general adaptability and of great value: 1. Instructions should be specific and definite to the night superintendent and head nurses to notify the house physician of any complaint of abdominal pain by the patient, of hiccough or vomiting, of a special rise of pulse or respiration, of sweating or of signs of collapse. 2. House physicians should note the character of the pain, as to (*a*) onset—whether only an aggravation of slight abdominal pain, such as is common both with constipation and with diarrhœa, or whether it was sudden, intense pain which caused the patient to call out, and which, though relieved by stupes and ordinary measures, soon recurred in paroxysms and grew worse; and (*b*) the locality—whether diffuse or localized in the hypogastric or right iliac regions; radiation, as to penis. It is to be borne in mind that abdominal pain of a severe character may be associated

[1] Sulla peritonite da propagazione dall'intestino nel corso della febbre tifoidea. Riforma med., 1900, p. 210. Centralblatt für innere Medicin, 1901, vol. xxii. p. 101.

[2] On Perforation and Perforative Peritonitis in Typhoid Fever. Philadelphia Medical Journal, 1901, vol. vii. p. 116.

with an acute pleurisy, with distended bladder, with cholecystitis, and with a packed rectum, or may follow an enema. 3. State of the abdomen—the conditions to be noted in writing at once as to the following particulars: (*a*) whether flat, scaphoid, or distended, and, if distended, whether the distention is uniform or chiefly hypogastric; (*b*) respiratory movements, whether present, if uniform, and seen both below and above the navel; (*c*) palpation, as to tension and pain, locality and extent, and degree of pressure necessary to elicit; muscle rigidity and spasm, whether present or not, and in which special locality, and noting particularly its absence or presence in the hypogastric region and the right iliac fossa; (*d*) percussion; character of note in front and in flanks; liver flatness, extent in middle, nipple, and in mid-axillary lines; note specially every third hour; remember, too, that obliteration may occur in a flat as well as in a distended abdomen; auscultatory percussion may be helpful; (*e*) auscultation; obliteration of signs of peristalsis; presence of friction; (*f*) examination of the rectum; whether tenderness or fulness between the rectum and bladder; (*g*) stools; character, frequency, presence of blood or sloughs. 4. General condition of the patient: (*a*) facies, whether change in expression; risus, slight or marked; pallor; sweating, etc.; (*b*) pulse, change in rhythm, rate, and force; (*c*) temperature, whether a drop or not; whether after a tub or not; (*d*) respiration, sudden increase not infrequent; whether shallow or sighing; (*e*) sweating, if subject to, during attack; if onset with pain; whether local or diffuse; (*f*) vomiting, whether with onset of pain or not; character of vomiting; (*g*) hiccough. 5. Blood-count, leucocytosis, whether stationary or rising; may be marked and early. In a majority of cases well followed there is a rise. The constant leucopænia in typhoid fever has to be taken into account. Also a count of the red blood-corpuscles and hæmoglobin, as a decided drop might indicate hemorrhage.

Considerable attention has been directed recently to the occurrence of typhoid bacilli in the urine of typhoid fever patients, to the occurrence of typhoid cystitis, and to the consequent necessity to disinfect the urine of all typhoid fever patients—Gwyn,[1] Curschmann,[2] Neufeld,[3] Schumburg,[4] Smith,[5] etc. With a view to prevent the occurrence of typhoid cystitis, to rid the urine of typhoid bacilli, and to limit the spread of typhoid fever, the internal administration of urotropin (7 to

[1] The Disinfection of Infected Typhoid Urines. Philadelphia Medical Journal, 1901, vol. vii. p. 80.

[2] Ueber Cystitis typhosa. Münchener medicinische Wochenschrift, 1900, No. 42.

[3] Deutsche medicinische Wochenschrift, December 20, 1900.

[4] Zur Disinfektion des Harns bei Typhusbakteriurie durch Urotropin. Deutsche medicinische Wochenschrift, 1901, No. 9.

[5] Ueber die echte typhöse Cystitis und ihre Behandlung. Monatsbericht für Urologie, 1901, vol. vi., Part 5.

15 grains three times daily) has been much recommended. In general this is a very valuable remedy, and it is usually well borne. It is well, however, to remember that several cases of hæmaturia following its use have been reported—Brown,[1] Griffith,[2] Gordon,[3] etc.

A number of cases of hemorrhagic typhoid fever have been reported recently by Stahl,[4] Musser and Sailer,[5] Hamburger,[6] Nicholls and Learmonth,[7] Dickinson,[8] Eshner and Weisenberg,[9] etc. Hamburger gives a good review of the literature of the subject. An unusual case of hæmoglobinuria complicating typhoid fever and terminating in recovery of the patient is reported by Musser and Kelly.[10]

DIPHTHERIA. McCollom[11] urges the use of large doses of antitoxin in the treatment of diphtheria, stating that, as we cannot estimate the amount of toxin generated by the diphtheria bacilli in the false membrane, we should administer the antitoxin in dose sufficient to produce the effect desired—that is, shrivelling of the false membrane, lessening of the amount and fetor of the nasal discharge, and improvement in the general condition of the patient. He says that when one sees a patient with membrane covering the tonsils and uvula, profuse sanious discharge from the nose, spots of ecchymosis on the body and extremities, cold, clammy hands and feet, a feeble pulse, and the nauseous odor of diphtheria, and finds that after the administration of 10,000 units of antitoxin in two doses the condition of the patient improves slightly; that after 10,000 units more have been given there is marked abatement in the severity of the symptoms; that when an additional 10,000 units have been given the patient is apparently out of danger, and eventually recovers—one must believe in the curative power of antitoxin. When one sees patients in whom the intubation tube has been repeatedly clogged, when the hopeless condition of the patient changes for the better after the administration of 50,000 units, one cannot help but be convinced of the importance of giving large doses of antitoxin in the very severe and apparently hopeless cases. In the majority of instances these large doses are not required, particularly if the patients are seen

[1] Hæmaturia following the Administration of Urotropin. British Medical Journal, 1901, vol. i. p. 1472.

[2] Ibid., p. 1617.

[3] Hæmaturia following the Use of Urotropin. American Medicine, 1901, vol. ii. p 848.

[4] Gangrenous Dermatitis Complicating Typhoid Fever. Transactions of the College of Physicians of Philadelphia, 1900, vol. xxi. p. 33.

[5] International Medical Magazine, 1899, vol. viii p. 827.

[6] Johns Hopkins Hospital Reports, 1900, vol. viii. p. 309.

[7] The Hemorrhagic Diathesis in Typhoid Fever, etc. Lancet, 1901, vol. i. p. 305.

[8] A Case of Hemorrhagic Typhoid Fever. Lancet, 1901, vol. ii. p. 24.

[9] Hemorrhagic Typhoid Fever. AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1901, vol. cxxi. p. 281.

[10] Hæmoglobinuria Complicating Typhoid Fever. Philadelphia Medical Journal, 1901, vol. vii. p. 119.

[11] A Plea for Large Doses of Antitoxin in the Treatment of Diphtheria. Boston Medical and Surgical Journal, 1900, vol. cxliii. p. 627.

early in the attack, 4000 to 6000 units being enough to produce the characteristic effect on the membrane. Dr. Osler[1] suggests that those in charge of institutions who still have any lingering doubts (concerning the efficacy of diphtheria antitoxin) should, in the interests of their little patients, and in a spirit of humility, visit the South Department of the Boston City Hospital, and learn a few salutary lessons from its director, Dr. McCollom.

REPORT OF A CASE OF FIBRINOUS BRONCHITIS, WITH A REVIEW OF ALL CASES IN THE LITERATURE.[2]

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Cases of fibrinous bronchitis, or plastic bronchitis, or bronchitis pseudomembranosa, or bronchial croup, are as interesting as they are rare. The following is the history of a case observed in the Maternity Ward of the Johns Hopkins Hospital in September last.

Maggie S., colored, aged twenty-two years; II.-para. Rather emaciated looking; very dull mentally.

Family History. No tuberculosis; father suffers from rheumatism; otherwise negative.

Past History. Labors normal. Menstrual history normal. Six years ago, in the fall, she had an attack similar to the attack observed in the hospital, the symptoms being cough, pain, respiratory distress, profuse expectoration, containing, evidently from patient's description, branching casts. Following this attack she had "pneumonia," being confined to bed one month. Every year in the fall since that time she has had similar attacks. These attacks had no relationship to her previous pregnancies, which occurred two and three years previous to entrance to hospital, respectively. (See Note I) She has lost in weight throughout the last two years. She entered the hospital in the ninth lunar month of her pregnancy, on July 27th. At the time of her entrance she had a temperature of 101.5°, which, in two or three days, had dropped to normal. From this time until the time of her delivery there is no note on her history of any abnormal condition, and for the last twelve days of this period she was under personal observation and nothing abnormal was noted. She was delivered on September 12th. She came to the delivery-room with a normal temperature, no cough, respiratory distress, or expectoration.

Present Illness. Her puerperium was normal except for a slight cough and a slight evening temperature—100° to 101°. She had the signs of a light bronchitis, lengthened expiration, and crepitant râles at the bases in the back.

On the tenth day she was allowed up in her chair, but the discomfort

[1] The Principles and Practice of Medicine, 1901, fourth edition, p. 155.

[2] Parts of this paper were read at the Johns Hopkins Medical Society on June 2, 1901, and specimens demonstrated.

from her cough increased so much that she was put to bed again, her evening temperature having touched 102°.

On the thirteenth day of her puerperium I was in the ward delivering another case, and my attention was called by the nurse to the patient's extreme dyspnœa.

Physical Examination (during first attack, observed on September 25th). Patient is sitting up in bed in intense dyspnœa. All the accessory muscles of respiration are called into play. The alæ nasæ dilate with each respiratory movement. She coughs incessantly, and brings up large masses of expectoration. She is complaining of an intense sharp pain radiating from the lower left side through the back, increased on coughing. Respirations are 35 per minute; pulse, 100; temperature, 102°. Loose tracheal râles are heard in the throat. On palpation there is normal vocal fremitus throughout. On percussion there is high tympany in both lower axillary regions, higher and approaching dull on the right. On auscultation over the fronts respiration is prolonged and accompanied by fine moist râles. The breathing is more intense in the right lower axilla, and approaches tubular breathing in its quality. The inspirations are here accompanied by râles distinctly crepitant in character. There is impaired resonance in the right lower back and harsher and louder breath sounds, and in one region covered by the bell of the stethoscope the breathing is distinctly tubular and accompanied by crepitant râles. The attack of intense dyspnœa lasted about one hour. The expectoration contained the large casts, from which I have prepared sections, and some smaller incomplete casts. Three days after the first attack Dr. Futcher made a careful physical examination, with the following result:

Right Lung. Percussion note and breath sounds are particularly clear throughout the entire front. The note is slightly impaired over the lower subscapular and axillary region. Over the lower interscapular and throughout the subscapular region the breath sounds are enfeebled, and there are a few moist râles on expiration. The voice sounds are slightly impaired.

Left Lung. On percussion the note is clear throughout the upper front and distinctly impaired throughout the lower half of the interscapular region. On auscultation a definite pleuritic friction rub is audible over the second, third, and fourth interspaces in the parasternal line, and is audible out to the anterior maxillary line. The breath sounds are not tubular. In the third interspace there is a cardiac respiratory rhythm to the friction rub. Between the mid-axillary and anterior scapular lines, and extending from the apex of the axilla to the costal margin, there is a definite area of impaired resonance, over which the breath sounds are almost inaudible and the voice sounds enfeebled. In the lower intrascapular region there are fairly numerous medium moist râles, some of which approach a crepitant quality.

Heart. Point of maximum impulse is best seen and felt in the fifth interspace 10 cm. from the median line. There is normal cardiac dulness. Both sounds are audible at the apex. The first is accompanied by a very soft systolic bruit. In the third interspace a pleuropericardial friction rub is audible as noted above.

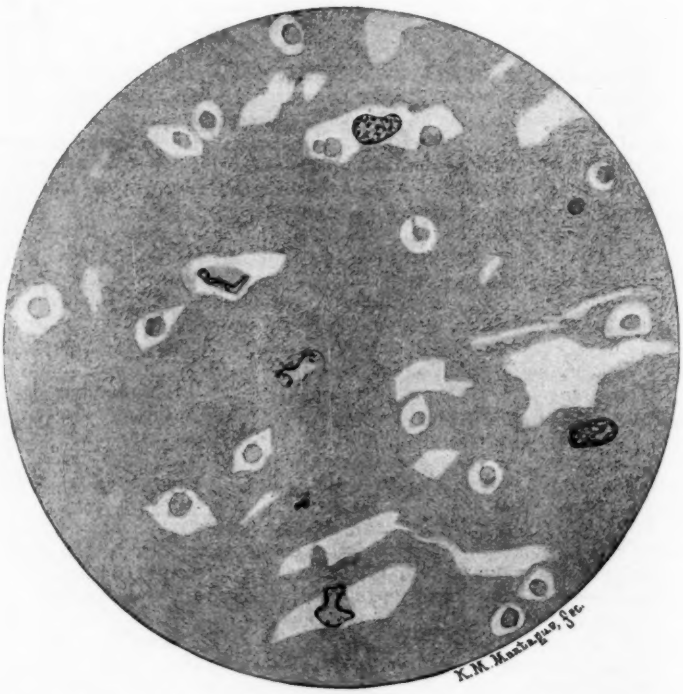
Leucocytes at this date were 5000, and the differential count was normal.

Further course of present illness: She had another paroxysmal

attack, not so severe in character, on October 6th (eleven days later), in which she coughed up the large cast which is represented in the drawing, and several smaller less complete casts. (See Fig. 1.) The sputum expectorated during this attack on October 6th showed the following characteristics: Amount, 100 c.c.; separates into three layers on standing: (1) a granular detritus; (2) milky fluid; (3) branching cast.

First Layer. Cells; alveolar epithelium, leucocytes which were not eosinophiles.

FIG. 1.



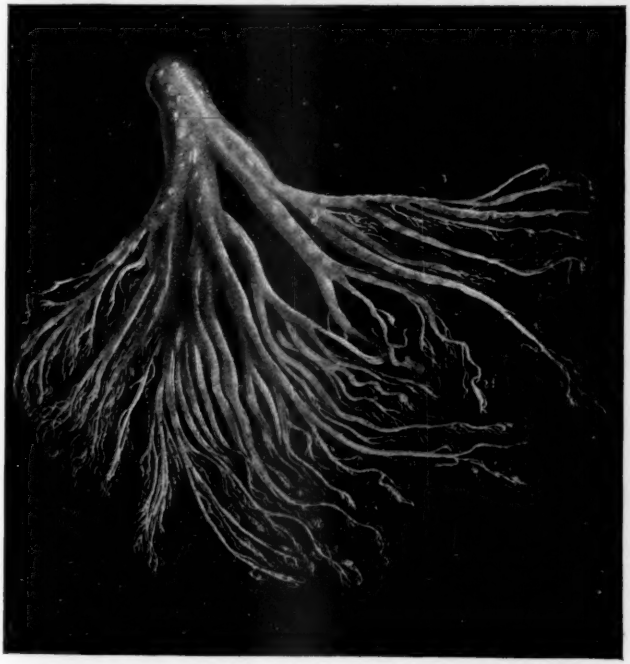
Second Layer. Bacteria, streptococci and diplococci; no tubercle bacilli, no Charcot-Leyden crystals, or Curschmann's spirals.

Third Layer. Branching casts which contained cells, the nature of which was not determined. No tubercle bacilli, no Charcot-Leyden crystals.

In the intervals between the attacks there was some slight respiratory distress and an evening temperature ranging between 100° and 101°. Only on a few occasions did the temperature reach 101°, so that a uterine culture was not taken, but puerperal infection could be definitely excluded by the absence of local signs. She had a slight cough, and occasionally expectorated small casts, which caused her very little dis-

comfort. On one occasion a to-and-fro vibrating sound was heard on expiration in the right axilla. This corresponds in character to the "ventilgeraüsch" of the Germans. The urine was negative throughout. The patient continued to have a slight cough and dyspnœa and slight evening temperature, and left the hospital in spite of our protests, October 10th. She remained in Baltimore for three weeks after her discharge from the hospital, and had frequent attacks of violent coughing and expectorated "tree-like things" (description of a friend). She then went to her home in Virginia, where she died two weeks later. No data as to the cause of death are obtainable.

FIG. 2.



Cast expectorated; three-fourths natural size. Drawn from fresh specimen.

Casts—Macroscopical Structure. The casts are of various sizes, the largest being 10 cm. long and showing branchings down to the seventh or eighth degree. (See Fig. 2.) They number about twelve. Their color is white, their consistency that of fibrin. Many show little intumescentia over the surface of the larger branches due to little bubbles of contained air. The morphology of the casts is best seen by looking at the illustration showing a portion of the cast embedded, from which the sections have been cut. (See Fig. 2.) It shows an outer stratified layer or skin, which encloses, in what would otherwise be a very large

lumen, various separate whirls and cylinders. One immediately gets the impression from looking at this cross section that the exudation starts in the finer bronchi, and is then gradually pushed up into the larger bronchi, where additional exudation is apposed. That is, supposing the largest branch of a large cast to represent a cast of the bronchus of the third degree, the substance forming that cast represents not only exudation formed in that bronchus itself, but also that formed in all the bronchi below the level of this bronchus of the third degree. Various portions of the cast were hardened in Müller's fluid or by the formalin alcohol method, and were stained by the following methods:

1. Weigert's fibrin stain (celloidin sections). 2. Hæmatoxylin and eosin (celloidin sections). 3. Eosin and methylene blue (paraffin sections) (see Wright and Mallory). 4. Ziel-Nielson's method for tubercle bacilli (paraffin sections). 5. Unna's method for mucin (paraffin sections).

Results. 1. A surprisingly small portion of the sections take Weigert's fibrin stain. The greater number of fibrin fibrils are distributed in the outer layer or "skin" of the cast, or in the outer layer of the smaller whirls. The stain brings out organisms, streptococci and staphylococci, generally adhering to the outer side of the cast, though a very few are occasionally found in the mucus in the inside of the cast.

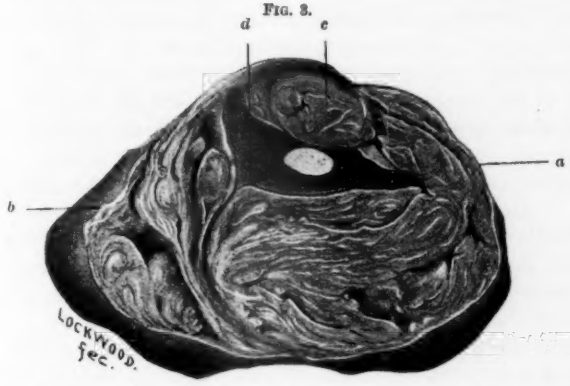
2. *Hæmatoxylin and Eosin.* This stain shows a ground substance consisting of two apparently distinct materials, a fibrillar substance taking a deep eosin stain, which is concentrically arranged about the lumina of the separate whirls. This encloses a second substance which is transparent and takes a light tinge of hæmatoxylin. Undoubtedly this latter substance, from its optical properties and from its staining reaction, is mucin. The substance taking the eosin stain so deeply is not altogether fibrin, but in part shows itself to be such, for it contains the fibrillæ which take the fibrin stain. What the nature of the portions that do not take the fibrin stain is it is difficult to say from the data at hand. Cells are very numerous, and are for the most part mononuclear leucocytes, though some polynuclears are seen. They are more numerous in the mucin ground substance than in the eosinophilic material. There are a few alveolar cells, many of which contain blood pigment; a very occasional red blood-cell is seen. Beside these cellular elements there are certain bodies which we supposed at first were cellular degenerations, but which in other stains took on a more specific character, and are, perhaps, some peculiar form of organism. These will be spoken of under 4. There were also a small number of cells showing eosinophilic granules. None of these are polymorphonuclear leucocytes, but are either degenerating epithelial cells or large mononuclear leucocytes.

3. *Sections stained in eosin and methylene blue.* These show the ground substance to be reticulated and taking altogether a deep eosin stain. The methylene blue stains only the nuclei of the cells. There are no mastzellen. This stain brings out well the few eosinophilic cells.

4. *The tubercle stain* showed no tubercle bacilli, but brought out those peculiar bodies which are mentioned above. They are irregularly round, from 7 to 15μ in diameter. (See Fig. 3.) They retain the fuchsin, keeping a deep red color. They have apparently a double

contoured outer shell, from which in certain places the deeply staining protoplasm seems to have shrunk away. Vacuoles of various sizes are seen in their protoplasm. The protoplasm stains diffusely, but shows a few more deeply staining granules. The shell seems to have a tendency to roll up on itself. These bodies were first seen in the sections stained for tubercle just four days before the reading of this paper at the Johns Hopkins Medical Society on June 2d, so that it is too early to give accurate descriptions of them or to judge as to their nature. The specificity of their staining and morphology suggests that they are some form of organism. After finding them here we looked for them in Weigert sections, and found that they retained Weigert's stain, showing the same characteristics with this stain as with the tubercle stain. They also take a specific stain in the eosin methylene blue method. In this they show well the double contoured, highly refractive outer shell, but

FIG. 3.



Drawn under a magnification of 16, showing the portion of the cast at the level of its primary division into three branchings. From the specimen embedded in celloidin, thus giving an idea of the cross section and the third dimension.

a and *b*. Outer skin. *c*. Separate whirl representing exudate from a smaller branch. *d*. Largest branching.

none of the details of the protoplasm are brought out. In the hæmatoxylin and eosin stain these bodies look like degenerated nuclei, retaining the hæmatoxylin.

5. Unna's method with polychromethylene blue for mucin shows a substance taking a blue stain and a more opaque substance taking a beautiful red.

Cultures were made from the inside of the main branch of the largest cast after searing its surface, and streptococcus pyogenes and staphylococcus aureus obtained in pure culture. No diphtheria bacilli or pneumococci were cultivated.

Collections of all the cases in the literature have been made by Valleix (1843), Peacock (1854), Lebert (1869), Biermer (1867), Riegel (1875), West (1889). Lebert's article is very complete. It is entitled

"Ueber das Vorkommen fibrinoser entzündungsproducte in den Bronchien und Lungen-alveolen—Ueber fibrinose oder Pseudomembranose Bronchitis und Pneumonie, Bronchitis Fibrinosa, Bronchitis Pseudomembranacea, Pneumonia fibrinosa."

He excludes from consideration all cases secondary to diphtheritic inflammation of the pharynx and larynx. These are cases caused, as we now know, by the Klebs-Löffler bacillus. They are especially discussed by Peter, Millard, and Trousseau. Lepine, who reviews this whole subject, gives only one authentic case, which he cites from Trousseau. One is led to expect that actual cases are rare, for the subject receives always at the hands of French writers general consideration. It is most often seen following tracheotomy. Lebert comes back to the term "fibrinous bronchitis" as opposed to Biermer's "pseudomembranous bronchitis." His group of cases of imperfect observations—thirty-two in all—are interesting mainly from an historical stand-point. Galen evidently observed a case and considered the expectorated masses vessels of the lung. Donatus (1586) describes the casts as "venæ frustum parvum per tussum excreatum e pulmonone." Nicol Tulpius as "surculi venæ arteriosæ expectorati." Bussiere (1750) made an autopsy on a case in all probability associated with tuberculosis, and found a cast *in situ*, thus proving that they were not vessels of the lung, but bronchial secretion. In the early part of the eighteenth century the casts were commonly called bronchial polypi: thus Samber (1727) and Ruyschius (1721). The most interesting case in this group and unique in the whole history of the disease is that of Hayn (1844), who describes finding at autopsy in an infant, aged three weeks, extensive casts throughout the bronchi of both lungs. Lebert's second group of cases—five in all—are not cases of fibrinous bronchitis. They are fatal cases of bronchopneumonia in which at autopsy bits of membrane are found in the bronchi, never forming any definite branching casts, but being scattered here and there throughout the pneumonic areas. Lebert's third group are cases of acute fibrinous bronchitis including seventeen cases. He gives a careful analysis of these cases, four of which were fatal, and complete histories of seven cases, including the four fatal ones. Three of these four were fatal in an attack, and one died in typhoidal collapse. The cases of Lebert's fourth group, associated with pneumonia, are, indeed, remarkable, and very suggestive. He calls attention to the fact well established through the researches of Lobstein, Remak, and Rokitansky that in lobar pneumonia little fibrinous plugs are expectorated in a certain proportion of cases (40 per cent. according to Remak), and are frequently found at autopsy in the smaller bronchi. He then reports five cases out of the literature in which definite branching casts of the bronchi were found in pneumonia. Four of these five cases were fatal, and in three out of these four casts

were never expectorated but were found at autopsy. All were fatal early in the disease. His fifth group of cases, including twenty-seven, are cases of idiopathic chronic fibrinous bronchitis. He gives a careful analysis of these, which it will be unnecessary to repeat, as the analysis of the cases given below covers the ground. There are seven cases in his sixth group of chronic symptomatic fibrinous bronchitis. Four of these are associated with the last stages of pulmonary tuberculosis; one with fatal abscess of the lung and mitral disease; one with syphilitic ulceration and stricture of the larynx, and one with empyema, this last case being the only one that was not followed to a fatal termination and autopsied.

I have tabulated all the cases that have been reported since the publication of Lebert's paper in 1869 in French, German, English, and American journals and monographs. Allow me to present an analysis of these cases. The cases reviewed fall naturally into the nine groups given below:

I. Chronic bronchitis with expectoration of branching casts of the bronchial tree: twenty-seven cases.

II. Acute bronchitis with expectoration of branching casts of the bronchial tree: fifteen cases.

III. Cases in which branching casts were not expectorated, but were found in the bronchi at autopsy: six cases.

IV. Cases in which the casts expectorated showed no dichotomous branching: eleven cases.

V. Expectoration of branching casts in organic heart disease: ten cases.

VI. Expectoration of branching casts in pulmonary tuberculosis: fourteen cases.

VII. Expectoration of small casts, often non-branching, in association with asthma: five cases.

VIII. Formation of casts in the bronchi in association with pulmonary œdema following thoracentesis: four cases.

IX. Cases whose classification is doubtful because of incomplete reports: six cases.

Analysis of Cases in Group I. *Etiology.* Sex: Males, 15; females, 11; (?) 1.

Age: First decade, 1; second decade, 3; third decade, 5; fourth decade, 9; fifth decade, 4; sixth decade, 2; seventh decade, 2; (?) 1. This shows progressive increase up to middle life, and thence a decline.

Occupation: There are five cases in which occupation is given as possibly having some bearing: Street, "captain of the hold" in a ship; Stirling, dock laborer; Schittenhelm, grave-digger; Roque, blacksmith; Hall, painter.

Infectious Diseases. There is a history of previous pneumonia in Worthington's case, and also in Singer's, Sax's, and Model's (Case

VII.) cases. In Caussaude's case there is onset after the third attack, with signs of massive pneumonia. In Roque's case influenza seemed to be etiological.

Chemical Irritants. In Schmidt's case the onset was after inhalation of smoke at a fire.

Family History. There is a family history of tuberculosis in Schittenhelm's and Beshorner's cases.

Personal History. An almost invariable past history is chronic bronchitis; duration varies much in different cases. In Brannan's case there was a history of angioneurotic œdema, and the author thinks that possibly the condition is the result of angio-œdema of the bronchi. In Mader's case there was a history of pemphigus previous to the onset of the attack, which condition extended to the mucous membranes of the mouth, throat, and larynx. The author is inclined to think that all cases represent a pemphigus of the bronchi.

Symptomatology. The onset is usually with an exacerbation of a chronic catarrh; some fever may accompany it. In Caussaude's case the onset was with a massive pneumonia at the left base. In Roque's case there was a long period which preceded the fibrinous expectoration, during which the patient had cough and night-sweats, and gradually became emaciated. A diagnosis of neurasthenia following influenza was made.

Duration—Interval Between Attacks and Duration of Attacks. The one most common characteristic in all the cases outside of the fibrinous expectoration is its tendency to recur in distinct attacks separated by intervals of freedom of longer or shorter periods. It is difficult to give the average duration of the disease, for, of course, many of the cases are lost sight of. Kisch's case is the longest in the history of the disease, lasting over a period of twenty-five years.

Dyspnœa. Paroxysmal attacks of dyspnœa generally immediately precede the fibrinous expectoration; but, strange to say, there are several cases in which the author distinctly states that there was very little dyspnœa: Singer, Model (Case VII.), Schmidt, Pousselt and Zenker (two cases). Evidently the respiratory distress is due entirely to bronchial obstruction. Between the attacks in which the larger complete branching casts are expectorated smaller ones or incomplete portions of casts are brought up, causing, as a rule, very slight dyspnœa. In some cases the expectoration of fragments of casts occurs over long periods before the complete casts appear: thus, in Regarde's case for a period of six years smaller non-branching cylindrical structures compared to macaroni were expectorated, causing considerable dyspnœa.

Cough. Paroxysms of coughing accompany the paroxysms of dyspnœa, and are proportionally intense, but in those cases in which the expectoration of casts is not associated with dyspnœa there may be

very little or no cough. Several authors mention that the simple "rauspern" was all that was necessary to cause the expectoration of casts.

Fever. In six cases the paroxysmal attacks of coughing and dyspnœa were associated with slight fever—in Zenker's (Cases I. and II.), Hall's Bernoulli's, Caussaude's, and Street's cases. The fever range was high. Fever may be said to rarely accompany the attacks. Possibly this might point to a non-inflammatory etiology of the disease in its chronic idiopathic form.

Hæmoptysis. In only two cases was hæmoptysis associated with the expectoration of casts—in Sokolowski's first case and Street's case. In the latter there is suspicion of pulmonary tuberculosis from the history, and only one hæmoptysis took place while under observation—a period of six months. In Sokolowski's case, however, the hæmoptysis was copious and usually accompanied the expectoration of casts, which took place daily for a period of nine months. In Schittenhelm's case the hæmoptysis began at the onset of a florid tuberculosis which complicated the fibrinous bronchitis, resulting fatally. Stirling's case on two occasions had hæmoptysis amounting to one ounce, but the author does not say whether there was any association with expectoration of the casts.

In Model's (Case VII.), Mader's, Regarde's, and Worthington's cases the casts were occasionally blood-tinged. Hæmoptysis may be said, then, to rarely accompany the expectoration of casts.

Physical Signs. It is impossible to make a diagnosis from the physical signs. Theoretically if the casts were *in situ* in the bronchi—that is, before the expectoration—the physical signs would be those of what the Germans call "bronchial stenose"—that is, impairment or absence of breath and voice sounds in an area of the lung that is obstructed, with no change in the percussion note. Impairment of resonance is often noted, however, in an area apparently involved, and is usually explained on the ground of collapse of the lung as a result of persistent bronchial obstruction. All types of râles are heard, from crepitant to the large, dry, sibilant râles. The most characteristic adventitious sound is the so-called "ventilgeraüsch" of the Germans, "bruit de drapeau" of the French. This is a very coarse, dry, clicking sound, apparently from its character, caused by the flapping to and fro of loosened portions of the cast with inspiration and expiration. Model, Beshorner, and Caussaude have noted it especially in their case before the expectoration of the casts. Localization of the sign is usually in the lower portions of one or both lungs. Chronic emphysema with its characteristic signs is often associated.

General Symptoms. In several cases a considerable degree of loss of weight and strength has been noted (Baumgarten, Championnière,

Street, Waldenburg, Sokolowski, and Roque). Baumgarten attributes the emaciation in his case to the withdrawal of alcohol. Sokolowski's is attributable to the frequent hemorrhages from the lungs. Undoubtedly, however, the emaciation is not an uncommon symptom, due in most cases to the very frequent and exhausting attacks of coughing and dyspnœa. Night-sweats without any discoverable tuberculosis have been associated in a few cases. (Roque and Street.)

Subjective Symptoms. A feeling of oppression and tightness in the chest is the rule, with complete relief on expectoration of casts. The patient can often tell from which portion of the chest the casts come by a peculiar subjective sensation in one region. (Schmidt and Caussaude.) Pain is not a common symptom, though it is marked in some cases.

Complications, Sequelæ, and Results. Complication with some form or other of skin disease have been noted frequently. Thus in Mader's case pemphigus of the skin and mucous membranes preceded the attack of fibrinous bronchitis. In Waldenburg's case an impetigo capitis was noted six months after the symptoms had disappeared. Street's case was complicated by herpes zoster over the shoulder, and later by an eruption of an impetiginous character over the forehead. During the prevalence of the eruption the patient was free from attacks of fibrinous bronchitis.

In three cases the patient was under observation at the time of death. Schittenhelm's case died as the result of florid tuberculosis. The occurrence of fibrinous bronchitis in the course of tuberculosis is common, as will be seen later. This is the only case, however, of tuberculosis supervening on an attack of fibrinous bronchitis. Sokolowski's case was not autopsied. He died as a result of emaciation and anæmia. Mader's case died six months after the last attack of fibrinous bronchitis, the cause being laryngeal stenosis due to cicatrization of a laryngeal ulcer and hypostatic pneumonia. In only one case (Sokolowski's) was a fatal termination the direct result of the disease.

Macroscopical Characters of Casts. In Schittenhelm's case the casts were 10 to 15 cm. long. In Pousselt's case the picture of a cast 16 cm. long is given; in Beshorner's case, 10 cm. long; Stirling's case, 3 to 4 to 6 inches long; Kisch's case, 3 to 8 cm. long; Bernouilli's case, one cast 10 cm. long with ten branches. Championnière gives an excellent picture of a cast 10 cm. long; Waldenburg's case 4 to 5 inches long. All authors who give accurate descriptions note the lumen sometimes in the larger, sometimes in the smaller branches. Championnière noted the separate whirls inside of an outer laminated layer. Dr. Osler examined the casts in Worthington's case and states that "each cast consists of a variable number of laminæ arranged concentrically, those in the centre being much folded and involuted." Most of the branches are cylindrical, some of the larger ones are flat.

Irregularities on the surface of the cast are often noted, and are supposed to be due to bubbles of contained air.

Microscopical Characters of Casts. In eight out of the twenty-seven cases no detail is given as to the microscopical structure of the casts. The usual description is of a fibrillar stratified ground substance containing in its meshes leucocytes. Pousselt, Schittenhelm, and Model are the only authors who state definitely what the characters of these leucocytes are, and they found them to be for the most part mononuclear leucocytes. Sokolowski, Kisch, and Stirling found red corpuscles. Schmidt finds peculiar bodies in the casts which he considers analogous to the rows of red blood-corpuscles, such as are seen in thrombosed capillaries. He also finds corpora lecithinoidea. Alveolar epithelial cells are commonly found, but no authors mention cylindrical epithelial cells, and in my case, after a careful search through many sections, I did not find one. No authors reporting cases in this group mention eosinophiles. In Model's (Case VII.) and Waldenburg's case there were very numerous fat-droplets throughout the casts, and in Model's case the milky appearance of the sputum led the author to suspect that his patient was fooling him. His conclusions with reference to his case are that it represents a lymphorrhagia or chylorrhagia, for (1) a chemical analysis of the sputum and casts gives the same percentage of fat as is found in chylous exudates; (2) the cells in the casts were mostly lymphocytes; (3) there was no dyspnœa or fever associated with the expectoration; (4) the remissions over long periods are exactly similar to those seen in lymphorrhagia or chyluria. He concludes generally that many cases of fibrinous bronchitis may represent a chylorrhagia or lymphorrhagia, for (1) it is in many cases secondary to disturbances of circulation; (2) the absence of epithelium in the casts and the intact mucous membrane found at autopsy point to this explanation.

Charcot-Leyden crystals were found in both of Zenker's cases. Five authors state definitely that they found none. Other authors do not mention these structures. We shall hear more of this in connection with the cases related to asthma.

Chemical Composition of Casts. Most authors simply state that the main mass of the casts were composed of laminæ of fibrin, being influenced wholly by the past views with reference to this and the laminated structure and general consistency of the casts. Championnière (1876) was the first to suggest that the casts were not composed of fibrin but of mucin. He says that microscopically it has not the reticulated appearance of fibrin "mais bien l'aspect uniforme et presque hyalin des matieres muqueses." It resists the action of acetic acid. He noted little irregular whirls enclosed by an outer lamina, which whirls he considered to be mucous moulds of the bronchial glands. Caussaude (1889)

came to the conclusion that the casts were composed of syntonin from the following data:

a. Are insoluble in lime-water; do not swell in water at a temperature of 45°; are soluble in acetic acid; are therefore not mucin.

b. Insoluble in sulphate of sodium and chloride of sodium and its deposit in very irregular whirls and circles, and the fact that it does not take the Weigert stain. It is, therefore, not fibrin.

c. Put in the incubator and immersed in a 1 : 1000 solution of hydrochloric acid at the end of twenty-four hours some solution had taken place.

The filtrate when neutralized gave a precipitate which redissolved on the addition of lime-water. Are, therefore, syntonin.

Stirling stated that they consisted of coagulated albumin, soluble in alkalies. Beshorner (1893) states that the casts are not fibrin, for lime-water dissolves them; they do not swell up in acetic acid, and, excepting a few fibrils, they do not take the fibrin stain. The fibrillæ that do take the stain look like the fibrillæ in the central thread of Curschmann's spirals. The author concludes that the exudate is like that in enteritis pseudomembranacea. Pousselt describes portions that take the fibrin stain enclosing mucus which does not take the stain, and is surprised that sections at different levels show portions morphologically similar, some of which take the fibrin stain and some of which do not. Schittenhelm concludes that chemically they are fibrin, for they digest in artificial gastric juice and decompose in hydrogen dioxide. The fibrin stain shows portions which take the stain and which enclose mucus in which are embedded cells.

Study of Bronchi at Autopsy. Schittenhelm's case was fatal as a result of florid tuberculosis. He could determine no especial localization of the tubercles about the bronchi. He found the alveoli and the bronchi of the right lower lobe filled with desquamated epithelial cells and fibrin. The bronchial mucous membrane showed nothing except dilated mucous glands which contained desquamated cells and mucus. The peribronchial tissue showed dilated capillaries infiltrated with leucocytes. Some of the bronchi of the right lower lobes contained plugs, but no definite branching casts. The fibrin stain gives fibrin fibrils throughout the tissue of the lungs, especially about the tuberculous area (but the author does not lay stress on this, as it is found in uncomplicated tuberculosis). Streptococci are seen free in the lumen and in the glands of the bronchi, but none are found in the tissues. No pneumococci, no diphtheria bacilli. He concludes that the disease is a result of a desquamative catarrh of the alveoli of the lobe of the lung, and an acute exudation into the bronchi, bronchioli, and alveoli.

Group II. The second group of cases, the acute cases with expectoration of branching casts, number fifteen.

Etiology. Sex: Males, 11; females, 3; (?) 1.

Age: First decade, three; second decade, six; third decade, one; fourth decade, four; seventh decade, one. There is no striking preponderance in youth.

Occupation: Occupation was deemed etiological in the following cases: Glasgow's case, a painter; Ott's case, a knife-sharpener; Sokolowski's case, a physician who was doing bacteriological work, and the author thinks it was a laboratory infection, as he cultivated streptococci and staphylococci from the sputum.

Acute Diseases. (*a*) *Pneumonia.* Koch's case, a man, aged eighteen years, had suffered three attacks of pneumonia, the last of which was characterized by great dyspnœa, a great deal of cough and expectoration, the latter containing numerous non-branching "gerinnsel." Temperature fell on the seventh day by lysis, and six days later the patient was seized with a violent pain and a paroxysm of intense dyspnœa and coughing, which was relieved by the expectoration of three or four branching casts. In Ott's case the expectoration of branching casts preceded the pneumonia; there was no expectoration of casts during the disease. He cultivated the staphylococcus pyogenes aureus and Fränkel's pneumococcus from the casts.

(*b*) *Typhoid Fever.* In Moller's case there was expectoration of casts coming on twenty-three days after the onset of so-called "typhoid." The diagnosis was perhaps doubtful.

In Souque's case, on the thirtieth day of a typhoid complicated by cutaneous abscesses which kept the temperature slightly elevated, there was a paroxysm of intense dyspnœa which was sufficient in duration to make tracheotomy necessary. No relief was experienced from this operation, and the tube was removed. From the tracheal wound a branching cast 15 cm. long was coughed up. There is no further history.

In this connection I might mention a case which I have not classed in any group, but which is both interesting and unique. This is the one reported by Eisenlohr. A typical case of typhoid with considerable bronchitis at the onset and expectoration, without respiratory distress or effort, of exquisitely branching casts on the second, third, fourth, and sixth days after the onset. The casts dissolved in lime-water and contained red blood-corpuscles, leucocytes, and alveolar and cylindrical epithelium.

(*c*) *Scarlet Fever.* (Adserson.) Scarlet fever followed by broncho-pneumonia preceded the fibrinous bronchitis by some months.

(*d*) *Measles.* (Yäger.) Onset two days after convalescence from measles.

Basedow's Disease. Fritsche reports a case in the course of Basedow's disease.

Symptomatology of the condition as concluded from twelve out of the fifteen cases is somewhat as follows. (We exclude Koch's case following pneumonia; Souques', in typhoid, and Vintras' case.)

Onset. Preliminary acute bronchitis in eight cases varying in duration; in Herzog's case it had existed four weeks before the expectoration of casts. The characteristic expectoration usually appears about the end of the first week.

Chills. In Bettelheim's case chill at onset, followed three days later by the expectoration of casts. In Kretschy's case the patient had a chill every evening up to the fifth day of the attack. He died on the eleventh day. The author thinks it represents an acute infection.

Glasgow's case: A chill just before the expectoration of casts.

Fever. The fever range is high, in Kretschy's case averaging 40° C. in the evening. This high fever preceded the expectoration of casts by five days. In Fritsche's case the temperature was 40° C. in the first attack.

Distinct Attacks. The disease shows the same periodicity that is exhibited in the chronic form, consisting of a number of distinct attacks characterized by fever, dyspnœa, cough, and expectoration of casts. In the interval between the attacks the patient is comparatively comfortable, though still febrile. In Bettelheim's and Herzog's cases there was only one such attack, but in the latter case it was preceded by a fever and catarrhal symptoms in the bronchi, lasting four weeks. In Kretschy's case there were only two attacks, occurring on the fifth and seventh days of the disease. In Glasgow's case there were two attacks with a long interval between the attacks, the second attack being marked by freedom from dyspnœa and great ease in expectoration. It is quite common in all cases to have fragments of casts expectorated for long periods before and after the characteristic expectoration. In Fritsche's case there were two attacks with an interval of four days. The paroxysms generally occur in the morning.

Pain. The pain in the thorax is usually very intense during attacks. A feeling of oppression in the chest exists between the attacks.

Hæmoptysis was found in only two cases—Bettelheim's and Herzog's.

Number and Macroscopical Structure of Casts. The casts are remarkably few in number. In Bettelheim's case only two complete casts were expectorated. In Kretschy's case only four (duration, eleven days, resulting fatally). Macroscopically there is no difference from those seen in the chronic form.

Microscopical structure does not differ from those seen in the chronic form. Koch found Charcot-Leyden crystals in the casts. Adserson, Ott, and Vintras state definitely that these structures were not present. Herzog, from staining reactions and chemical tests, concluded that the casts were fibrin. Koch found that most of the cells were mononuclear

leucocytes. In Vintras' case the casts dissolved in lime-water. Souques thinks they were fibrin from their morphological appearance, but states that they did not give the fibrin reaction.

Result. Naturally some of the cases in this group might very well be the first attack of a condition which was to become chronic. Bettelheim's and Glasgow's cases suggest this, for after the subsidence of the acute attacks they had slight attacks for a month or so. In several of the chronic cases the first attack is very like in symptomatology to the acute form, and undoubtedly there are transitions between the two which might well be classed as subacute.

In the fifteen cases here considered four were fatal and three autopsied.

Autopsies. 1. Kretschy's case. Young man. Cast found *in situ* in right middle and upper lobes. The mucous membrane of the bronchi of one lung showed loss of epithelium and infiltration with white cells. The other lung showed œdema.

2. Yäger's case. Child, aged five years, died, the expectoration of casts diminishing with increasing dyspnœa and aphonia. Section showed a fresh adhesive pleuritis and pericarditis—bronchopneumonia of the left lung—a weak, incompletely adherent deposit in the larynx, which became thicker and more solid as bifurcation was approached, and extended into all the branches of the bronchi of the left lung and a little way into the right. The case followed an attack of measles. In its autopsy finding and clinical course it is almost identical with Hilton Fagge's case, which we have quoted above.

3. Vintras' case is unique in the history of this disease. An emaciated soldier, aged sixty-eight years, had suffered from cough for three months before admission. He had copious hæmoptysis a week before admission. Four days before admission an attack of intense cyanosis and dyspnœa relieved by expectoration of a branching cast. Seven days later another attack, and fifteen casts were coughed up. Seven days later the patient died in another attack.

At autopsy a rapidly growing epithelioma was found in the œsophagus opposite the bifurcation of the trachea, which latter would hardly admit a goose-quill. A cast was found projecting from the right bronchus into the trachea and obstructing the already strictured lumen of the latter. One small cast was also detected in the tubes of the right middle lobe.

Group III. The next group of cases includes those cases in which casts were found at autopsy, but were not expectorated during life. They number six. Clinically they do not belong to the group of fibrinous bronchitis. The probability is that many such cases go undiagnosed because no autopsy is made. Hutchinson's case is probably a case of diphtheria of the larynx in which extension of the membranes into the bronchi followed tracheotomy.

The two cases reported in Virchow's *Archiv* (1879) followed rupture of caseous lymph glands into the bronchi, both in very young children. Escherich's case was a fatal case of double pneumonia. Mazotti's case, a woman, aged seventeen years, died at the end of two weeks with high fever and dyspnœa. At autopsy the lesions of typhoid were found and a fibrinous inflammation of the bronchi. (This makes in all four cases showing an association with typhoid.)

Cutler's case is the only idiopathic case in the group. A woman, aged sixty-five years, had had acute bronchitis for three days; intense dyspnœa and cyanosis supervened, resulting in death in three hours. Section showed "pharynx and larynx healthy. A croupous membrane lay reflected on itself over the entrance of the two primary bronchi. When laid in place this membrane reached more than half-way up the trachea. Downward it extended into the minutest divisions of the bronchi, in many of them forming an almost solid plug. The alveoli contained no solid matter, and except in a few places where there was œdema and collapse they contained air." Evidently in these cases death occurred from asphyxia, because the casts were not expectorated. In this connection the rather bad prognosis of the disease in the two extremes of life might be mentioned, possibly having some relation to the fact that expectoration is difficult at these periods. A study of the cases in this group and the fatal cases of the second group shows the following facts: Out of twenty-one cases ten were fatal.

In the second group, Möller's case, aged thirteen years, not autopsied. Kretschy's case, a boy, autopsied; cast in bronchial tree. Yäger's case (measles), aged five years, autopsied; cast in bronchial tree. Vintras' case (epithelioma), aged sixty-eight years, autopsied; cast in bronchial tree.

In the third group, Cutler's case, aged sixty-five years, autopsied; cast in bronchial tree. Mazotti's case (typhoid), aged eighteen years, not autopsied; associated with typhoid. Escherich's case (pneumonia), aged twenty-seven years, autopsied; casts in bronchi. Virchow's *Archiv* (rupture of bronchial lymph gland), two cases, aged four months and four years respectively, autopsied; casts in bronchi. Hutchinson's case (diphtheria), child, autopsied; casts in bronchi and trachea.

Group IV. The fourth group of cases includes all those in which, although no definite arborescent moulds of the bronchial tree were expectorated, fibrinous structures evidently emanating from the bronchi were: eleven cases.

The symptomatology in some of the cases is very much like the symptomatology of the cases in the first and second groups. The difference is, perhaps, only a matter of degree, in this group the exudation being less in amount, perhaps more limited in area, and the expectoration taking place before a definite branching cast is formed.

In going over these cases one is impressed with the fact that the authors in many instances have reported in the same article cases belonging to the first and second groups. Thus have Chvostek, Kretschy, Model, Sokolowski, and Chauffaud. The natural inference is that these authors, having observed one case in which the expectoration contained these wonderful arborescent casts, kept their eyes open for other similar cases. It is well to separate these cases from those in the other groups to bring out the frequency of the cases in which arborescent casts are expectorated, or, rather, the infrequency. It might be mentioned that fibrin is frequently present in the expectoration in ordinary bronchitis. I have a specimen of a plug expectorated in a chronic bronchitis showing quite a marked content in fibrin (Weigert's stain). There are eleven cases in the group.

GROUP V. The fifth group of cases includes all those associated with organic heart disease—ten cases. There is no good reason why the cases in this group should not be included in the two groups of idiopathic cases. Heart lesions predispose to bronchitis through congestion. If this bronchitis assumes a special type there is no reason to suppose that the heart lesion is the etiological factor. We certainly consider the bronchitis of heart disease as an idiopathic bronchitis, though recognizing that a predisposing factor lies in the organic heart lesion. In Bernouilli's, Degen's, Lawrence's, Hint's, and Habel's cases the condition was chronic. It is worthy of note that all these cases were afebrile, and in three of them dyspnœa was absent during the attacks. This might indicate that the process was purely mechanical, and not inflammatory, the exudate taking place quickly and as a result of congestion. Seven of the cases were under observation at the time of death. In Degen's case, in a boy, aged sixteen years, the duration of the disease had been eight years; the autopsy findings were adherent pericarditis externum with exudation, aortic and mitral insufficiency; brown induration of the lungs. The right main bronchus and its branches contained a cast *in situ*, also the bronchi of the left lower lobe. Rohr's case: a woman, aged twenty-one years, under observation during the last month and a half of life, during which time she had loss of compensation and three attacks of fibrinous bronchitis. At autopsy: mitral insufficiency, infarct of lung, no casts, a tubercular kidney.

Beshorner's case; a male, aged forty-nine years. No clinical history. At autopsy a pleuritic exudation, adhesive pericarditis, nephritis, and peribronchitic infiltration of the lungs. It is worthy of note that in three out of the five cases that were autopsied adherent pericarditis was found. In Habel's, Hint's, and Schittenhelm's cases (all of which were under observation at the time of death), when compensation was lost, there were no longer any attacks of fibrinous bronchitis. Schittenhelm's case resembled an acute fibrinous bronchitis, the other two were chronic.

Structure of Casts. The four authors who investigated carefully found that the casts were not in the main composed of fibrin. Grandy and Beshorner found that the casts did not take the fibrin stain at all. Habel found a few fibrillæ staining for fibrin. Hint concludes that it was mucin not fibrin on rather insufficient data. No authors mention Charcot-Leyden crystals or eosinophiles.

Study of Bronchi at Autopsy. Grandy made a very careful study of the condition of the bronchi and of a cast *in situ*. He found dilatation of the vessels of the bronchi, clumps of leucocytes especially about the bases of the mucous glands, which latter were much dilated and filled with mucus, and it could be seen that this mucus merged into the casts. There were very great numbers of goblet cells present. The character of the staining reaction of the mucus was the same as that of the outer layer of the cast (blue with Van Gieson, red with thionin); but in the inner part the reaction had changed somewhat, showing that the mucus had altered in character.

Heart Conditions in this Group. Mitral insufficiency in five cases; mitral insufficiency and stenosis in one case; mitral stenosis in one case; mitral insufficiency and adherent pericardium in one case; adherent pericarditis in two cases.

Group VI. The sixth group includes those cases associated with tuberculosis of the lungs. There are fourteen in all, ten of which were under observation at the time of death, and were autopsied.

The expectoration of casts generally occurs late in the disease. In Homolle's case two casts were expectorated, one eight months before death and one eleven days before death. In Fraentzel's case casts were expectorated for the last seventeen days of life. In Pramberger's case a cast was expectorated fifty-five days before death and one eleven days before death. Jaccoud's case just one day before death expectorated two casts. Model's case (Case IV.) expectorated casts for two months before death. Model's case (Case V.) for ten days previous to death. Sokolowski's case, thirty days before death expectorated one cast. Casts were found at autopsy *in situ* in three cases—Homolle's, Model's (Case V.), and Klein's cases.

Model found a caseous infiltration of the walls of the right upper bronchus in which a cast was found *in situ*. Magniaux found peribronchial miliary tubercles in both apices, with denudation of epithelium and infiltration with leucocytes.

Structure of Casts. The casts are very few in number, and are the typical large branching structures found in the idiopathic form. All authors in this group state on rather insufficient evidence that the casts are composed of fibrin. Klein is the only author who goes carefully into the composition, and he finds that it does not take the fibrin stain and that it dissolves in chloroform. No authors mention staining sections of the casts for tubercle bacilli, so that if these peculiar bodies

which we have described in our cases were present they were overlooked. Tubercle bacilli were always found in the sputum.

All possible tuberculous conditions of the lung are found in this group of cases. Magniaux cultivated the diplobacillus of Friedländer from the blood, organs, and casts.

GROUP VII. Occasionally in the literature on bronchitis fibrinosa there is a reference to the relation of this disease to asthma. The suggestion of a relationship is brought about by the fact that there is a group of cases in which small fibrinous structures are expectorated along with the common contents of asthmatic sputum, Charcot-Leyden crystals, Curschmann's spirals, and eosinophiles. Are these cases to be classed as asthma or as bronchitis fibrinosa?

Schmidt has made elaborate studies into the sputum in asthma, and he found more or less large fibrinous structures in six out of eight cases of asthma which he investigated, using Weigert's stain to demonstrate the fibrin. He gives two cases especially in which the "gerinnsel" were quite large—2 to 3 cm. long. He describes them as "fädige und flocksige gerinnsel," which were composed for the most part of fibrin, and also contained some mucus in spiral arrangement, many eosinophilic cells, and numerous crystals. One of these cases was associated with hypertrophic rhinitis, and suffered typical asthmatic attacks. His conclusion is: "Berucksichtigt man aber, das wiederholt auch bei fibrinöser Bronchitis Spiralen gefunden worden sind, so durfte es in der that schwer sein, Fälle wie diesen, welcher dem Sputum nach eher zur fibrinöser Bronchitis, dem klinichen Bilde nach zum Asthma gerechnet werden muss, zu classificieren." The author also found small fibrinous structures in two cases of ordinary bronchitis. He concludes that the exudation of fibrin is quite common in inflammatory conditions of the bronchioles. (My preparation under the microscope is a plug from a case of ordinary bronchitis in emphysema. It shows fibrin in fair amount. Demonstrated at the Johns Hopkins Medical Society, June 2d.) Pousselt makes an elaborate analysis of many cases of bronchitis fibrinosa from the point of view of the relation of this disease to asthma. He concludes that the cases reported as fibrinous bronchitis can be classified into two distinct groups:

(*a*) "One in which the casts consist almost exclusively of fibrin containing very few cells and little or no mucus, and in which the expectoration takes place without any asthmatic attacks or the least subjective inconvenience. A relationship to any other pathological condition is not demonstrable. The etiology is obscure. There are very few cases of this essential primary fibrinous bronchitis. The course is chronic, with subacute, rarely acute attacks, in which casts are expectorated."

(*b*) "Second group. Very much more numerous than the cases of the first group. There are cases in which the fibrinous bronchitis is complicated by an asthma in which the casts are rich in cells and crys-

tals, and spirals are often found. The casts are also rich in mucus. The course is much more severe and acute." I will not tire you with the gross misstatements and contortions of fact upon which this classification of Pousselt's is based. They are numerous, occupying about one hundred pages. His other conclusions are that bronchitis fibrinosa is often complicated by asthma, but that the two processes are entirely distinct—the former not being in any case an extension of the asthmatic exudation into the larger bronchi. However this may be, the fact remains that there is a well-marked group of cases in which the so-called "asthma-eueger," Charcot-Leyden crystals, Curschmann's spirals, and eosinophilic cells are expectorated along with small fibrinous structures representing small casts of the finer bronchi, but generally not branching. The amount of dyspnœa in the attacks in these cases is much greater than the size and number and form of the casts would indicate if one looked to *bronchial* obstruction as the etiological factor in causing the dyspnœa. One is, therefore, tempted to classify these cases as asthma in which the exudative process has extended beyond the bronchioles in some areas of the lung, causing the formation of casts, generally not branching in some of the smaller bronchi. "Leyden has stated that one might consider asthma as a fibrinous bronchiolitis which occasionally can extend to the large bronchi, so that large 'gerinnsel' are expectorated. Nevertheless, one cannot identify asthma with fibrinous bronchitis, for there are many cases of the latter affection which have nothing to do with asthma. The cases which are related to asthma are characterized by the fact that the casts are filled with Charcot-Leyden crystals. Fränkel agrees with Leyden as to the relationship of the two diseases, but does not think asthma should be designated a fibrinous bronchiolitis, for the exudate is not for the most part fibrin." (From Pousselt.)

In going over the cases since 1869 I find that in five out of the seven cases in which Charcot-Leyden crystals have been found in the casts the latter have been small, generally possessing very few or no branchings. I am, therefore, tempted to place these in a group by themselves, showing a transition between fibrinous bronchitis and fibrinous bronchiolitis (asthma). They certainly represent a group distinct from the first and second groups, in which the casts are very large and show exquisite and numerous dichotomous branchings.

In Vierordt's case Curschmann's spirals appeared after the fibrinous structures had disappeared, thus suggesting the alternate occurrence of asthma and fibrinous bronchitis.

I am fortunate in being able to present, through the kindness of Dr. Osler, a case which I think represents this group well, and will bring in marked contrast the case spoken of earlier in the paper.

Kate M., aged forty-five years, white; first admission to hospital March 6 to 15, 1900. Complaint: Cough for several years, and lately some smothering sensation on lying down.

Family History. Father died of "asthma." Mother died of dropsy. Two sisters died of consumption.

Personal History. Has had eight children. Had a severe attack of rheumatism eight years ago. Is frequently a sufferer from tonsillitis. During the last sixteen years Dr. Mackenzie has removed about twenty polyps from the nose.

Present Illness. Has suffered from cough for periods throughout the last twenty years. The cough comes on in paroxysms at night, and is associated with attacks of shortness of breath. She says she can bring on a paroxysm by spraying her nose. She brings up considerable expectoration during these attacks.

Physical Examination shows typical signs of mitral insufficiency, with good compensation. Herpes about the lips and nose; tonsillitis while in the hospital; no note on the sputum; trace of albumin in the urine; hæmoglobin, 68.7 per cent; no temperature.

Second admission, April 15th; June 2, 1901. Complains that she has never been free from cough since she left the hospital. For the last two months this cough and "smothering feeling," as she calls it, have been worse.

Present Illness. Has been kept in bed on account of her heart lesion ever since entrance to the hospital. During this time she has had frequent attacks of dyspnœa and coughing and considerable amount of expectoration. No fever has been present. The attacks usually occur in the early morning. The respiratory distress is extreme, and vomiting is often associated with the expectoration. In the interval between these paroxysmal attacks she has, as a rule, been free from dyspnœa, though at times she has suffered considerably from it, having to remain in a reclining position in bed.

Physical Examination shows the signs of a chronic bronchitis; a well-marked mitral insufficiency; no signs of loss of compensation; hæmoglobin, 47 per cent.; no albumin in urine.

Sputum. Sputum contains Curschmann's spirals in abundance, eosinophilic cells, no tubercle bacilli. There are a fair number of very small branching casts, none over 3 cm. long, the majority averaging not over 1 cm. in length and only showing one or two incomplete branchings. Curschmann's spirals are seen at the end of the finer branches very frequently, and suggest the relationship spoken of above between bronchiolitis fibrinosa (asthma) and bronchitis fibrinosa. The calibre of the largest cast is not over 2 mm., and no lumen is seen on cross section. The casts consist for the most part of fibrin (as shown by Weigert's fibrin stain), containing leucocytes, generally polynuclear eosinophiles. No Charcot-Leyden crystals are seen. Sections cut longitudinally through the branches which end in Curschmann's spirals show that the fibrin from the casts is continuous with the fibrin in the central thread of the spiral. Streptococci and staphylococci are numerous in the casts.

GROUP VIII. The eighth group of cases includes those associated with acute œdema of the lung after thoracentesis. There are four such described by Ortner. In two of the cases fibrinous casts were expectorated (Hampeln and Ortner's second case), associated with a great amount of albuminous expectoration. In Ortner's case only one cast 3 cm. long was expectorated. The patient, a woman, died two months

later with increasing loss of compensation; adherent tuberculous pericarditis, double tuberculous pleurisy, œdema of the lungs, congestion of the liver, and ascites were found. At autopsy there were no casts in the bronchi. Ortner and Scriba describe cases which resulted fatally in which casts were not expectorated, but were found *in situ* at autopsy. In Ortner's case, in the right lung; in Scriba's, in the left, corresponding to the side on which thoracentesis was performed. In Ortner's case the autopsy findings were adherent pericarditis, mediastinitis, congestion of the liver, and ascites. In Scriba's case tuberculous pleurisy and pericarditis were found at autopsy. In this connection it is interesting to recall that in three out of the five cases of fibrinous bronchitis associated with heart disease which were autopsied, adherent pericarditis was found; also in one case in the second group adherent pericarditis was found.

GROUP IX. The ninth group includes a few cases which I have been unable to classify through dearth of description. (See References.)

In going over the literature I came across an interesting and unique case reported under the title of "Bronchite Pseudomembraneuse Aspergillaire Primitive," by Devillers et Renon, in *La Presse Médicale* for 1899. It affected a woman, aged forty years, who coughed up for a considerable time greenish membranes emanating from the bronchi, entirely composed of mycelium of aspergillus fumigatus. Her profession consisted in selecting seeds for cultivation. Her method of testing them was to feel their consistency with her teeth. It is supposed that she thus acquired this peculiar infection. Attacks of dyspnœa and cyanosis preceded the expectoration.

Expectoration of blood-casts of the bronchi following hæmoptysis is rather rare, and in consulting the literature I came across only three cases the references of which are given.

NOTE I.—*Note with Reference to the Relation of Pregnancy and Menstruation to Fibrinous Bronchitis.*

No such relation was made out in the case of Maggie S. Schnittzler (1864) observed a case. A woman after her sixth labor, instead of having her menstrual flow at the eighth week (as was her custom), had an attack of fibrinous bronchitis. For several months this phenomenon recurred. After her seventh and eighth labor the author made the same observation. This same case was again observed by Brik seventeen years later, when the woman was fifty-nine years old, in an attack of fibrinous bronchitis. Evidently at this age neither pregnancy nor menstruation could have had an etiological influence.

Pousselt quotes Stofelln, who observed a case in which attacks of fibrinous bronchitis replaced menstruation.

NOTE II.—*The Bacteriology of Fibrinous Bronchitis.*

In the cases considered in this paper the following bacteriological facts have been elicited:

GROUP II. Souques demonstrated streptococci, pneumococci, and staphylococci in the casts. Ott cultivated the staphylococcus aureus and the pneumococcus from the casts. Herzog demonstrated staphylococci and bacilli in the casts. Koch found bacteria which retained the stain by Gram's method always enclosed in mucus. He therefore does not consider them etiological.

GROUP IV. Sokolowski cultivated the staphylococcus albus and aureus from the sputum—a fact having no bearing whatsoever.

GROUP IX. Sokolowski demonstrated staphylococcus and streptococcus and a tetragenous conus in the casts. Neither diphtheria bacilli nor tubercle bacilli have ever been demonstrated in the casts.

Landireux and Triboulet (reference given under "Foreign References not Read") cultivated pneumococcus from the casts. It is an accepted fact that staphylococcus, streptococcus, and pneumococcus are etiological in the production of simple purulent bronchitis, so that their presence in the casts does not wholly explain the etiology of this rare disease. One observer has gone so far as to claim that in a case of his antistreptococcic serum was curative. Hoffmann (Nothnagel's *Handbuch*, xiii. Bd., iii. Th., 1 Abth.) recognizes two organisms as etiological: the pneumococcus and the diphtheria bacillus. He admits that the pneumococcus can be demonstrated in simple purulent bronchitis, but ventures the conjecture that cases in which it is alone or principally active develop fibrinous bronchitis; whereas, the purulent form appears where streptococci or other cocci overgrow it. This is only a conjecture.

Pinhini isolated a bacillus in three cases which, inoculated into the trachea of rabbits, practically reproduced the disease. Inoculation into other parts of the body was with negative result. His three cases occurred in three bricklayers who were working in the neighborhood of a sewer, and are supposed thus to have acquired the infection. I have not read the original article, "Contributio allo studio della Bronchite fibrin, acuta prim," in the *Riv. clin. arch. ital. di clin. med.*, 1889, p. 105.

COLLECTIONS OF CASES.

For all references previous to 1869, see Lebert's article. References follow the text, being arranged in groups.

GROUP I. 27 Cases. *Idiopathic Chronic Fibrinous Bronchitis: Branching Casts.*

1869. Waldenburg. Berliner klin. Wochenschrift, S. 208.
1869. Baumgarten. Medical and Surgical Journal of St. Louis.
1876. Championnière. Paris Thesis on "Bronchitis Pseudomembranosa."
1876. Worthington. Canada Medical and Surgical Journal.
1876. Zenler. Deutsch. Arch. f. klin. Med. (2 cases).
1877. Pernouilli. Deutsch. Arch. f. klin. Med., Bd. xx.
1877. Hall. St. Bartholomew's Hospital Reports for 1877.
1880. Street. American Journal of the Medical Sciences, vol. lxxix.
1882. Mader. Wiener med. Wochenschrift, No. xii.
1885. Bamberger. Allg. Wiener med. Zeitung, p. 155.
1886. Singer. Prager med. Wochenschrift, Bd. xi. S. 249.
1886. Sax. Wiener med. Presse, No. i., iii.

1837. Regarde. Thèse de Berne, "Contr. à l'Étude de la Bronch. Fibrineuse."
1888. Kisch. Prager med. Wochenschrift, No. 9.
1889. Caussade. Bull. de Soc. Anat., p. 371.
1889. Stirling. Practitioner, vol. xlii. p. 403.
1889. West. Practitioner, vol. xliii.
1890. Roque. La Presse Médicale. (Read in Dejean's Thesis.)
1890. Model. Ueber Bronchitis Fibrinosa, Case VII.
1893. Beshorner. Volkmann's Sammlung f. innere Med., No. 73.
1895. Sokolowski. Deutsch. Arch. f. klin. Med., vol. lvi. S. 476.
1896. Brannan. Medical News, vol. lxix. p. 169
1899 Chauffard. Rev. Internat. de Méd. et. de Chir.
1899. Schmidt. Centralblatt f. Allg. Path. u. Path. Anat., x. 425.
1900 Pousselt. Beiträge z. inn. med. Festsch. f. d. Cong. f. inn. Med., Wien., 1899–1900.
1900. Schittenhelm. Deutsch. Arch. f. klin. Med., S. 340.

GROUP II. 15 Cases. *Acute Fibrinous Bronchitis: Branching Casts.*

1870. Roth. Deutsch. Arch. f. klin. Med.
1873. Bettelheim. Casuistiche Mitth. über B. Fibrinosa, Wien. (Schmidt's Jahrbücher, vol. clxiii.)
1873. Kretschy. Wiener med. Wochenschrift, Nos. 14, 15, 16.
1876. Hajeks. Wiener med. Presse.
1879. Glasgow. Transactions of American Medical Association for 1879.
1883. Adserson. Hosp. Tidende, 3 R. i. 49–50. (Schmidt's Jahrb., vol. cciv.)
1882. Yäger. Würtemberger Aerztliche Correspondenz-Blatt, No. xv.
1884. Möller. Hosp. Tidende, ii. 12. (Schmidt's Jahrb., vol. cciv.)
1892. Koch, R. St. Petersburger med. Wochenschr., No. ix.
1893. Fritsche. Read in Schmidt's Jahrbücher, Bd. ccxxxvii. S. 219.
1897. Allen. Transactions of Medical Society of District of Columbia.
1898. Patton and Herzog. Journal of American Medical Association, vol. xxx. p. 25.
1900. Ott. Deutsch. med. Wochenschrift, xlvii.
1900. Vintras. Lancet, Sept. 15, p. 809.
1900. Souques. La Independence Médicale, Jan. 17.
Case in the course of typhoid fever.
1876. Eisenlohr. Berliner klin. Wochenschrift, No. xxxi.

GROUP III. 6 Cases. *Cases in which casts were found in bronchi at autopsy, but were not expectorated.*

1874. Hutchinson. Trans. of Path. Soc. of Phila. (Probably diphtheritic.)
1879. (?) Virchow's Arch. f. Path. Anat., Bd. lxxii., S. 294 (2 cases).
1883. Escherich. Deutsch. med. Wochenschrift, No. viii.
1884. Mazotti. Bolle delle Scienze Med. di. Bologna, June 14, 1884. Read in Centralblatt f. klin. Med. for 1885.
1887. Cutler. Boston Medical and Surgical Journal, p. 443.

GROUP IV. 11 Cases. *Cases in which the casts expectorated showed no dichotomous branching.*

1875. Chvostek. Wiener med. Presse.
1882. Kingsley. St. Louis Courier of Medicine, March.
1882. Madigan. Chicago Medical Review, p. 184.
1883. Kretschy. Deutsch. med. Wochenschrift, No. viii.
1881. Barron. The Lancet.
1889. Gibson. Practitioner, vol. xliii.
1890. Model. "Ueber Bronchitis Fibrinosa." Dissertation, Freiburg.
1893–94. (?) The Hospital, London, p. 240.
1896. Sokolowski. Deutsch. Arch. f. klin. Med., p. 482.
1898. Lepine. Rev. de Méd., 1898, vol. xviii. p. 835.
1899. Chauffarde. Rev. Internationale de Méd. et de Chir.

GROUP V. 10 Cases. *Expectoration of Branching Casts in the Course of Organic Heart Disease.*

1877. Bernouilli. Deutsch. Arch. f. klin. Med., Bd. xx.
1878. Degen. Würtemberger med. Correspondenz-Blatt, Bd. xlviii.
1886. Stack. Berliner klin. Wochenschrift, S. 221.
1892. Rohr. Correspondenz-Blatt f. Schweizer Aerzte, vol. xxii. p. 767.
1893. Lawrence. Lancet, vol. i. p. 247.

1893. Beshorner. Volkmann's Sammlung f. inn. Med., No. iii.
1897. Grandy. Centralblatt f. Allg. Path. u. Path. Anat., vol. viii. p. 513.
1898. Habel. Centralblatt f. innere Med., 1898, No. 1.
1898. Hints. Wiener med. Wochenschrift, vol. xlviii.
1900. Schittenhelm. Deutsch. Arch. f. klin. Med., p. 338.

GROUP VI. 14 Cases. *Expectoration of Branching Casts in the Course of Pulmonary Tuberculosis.*

1870. Tuckwell. Trans. of Path. Soc. of London (doubtful).
1874. Flint, Austin. Med. Record, vol. ix. p. 41.
1875. Homolle. Bul. de la Soc. Anat., t. x. p. 542.
1878. Fräntzel. Charité Annalen, v. Jahrgang, p. 295.
1881. Pramberger. Mittheil. d. Verein d. Aerzte in Steienmark, 1880, Graz, 1881, vol. xvii, pp. 3–68.
1884. Möller. Hosp. Tidende, vol. ii. p. 12. (Read in Schmidt's Jahrb., vol. cciv.)
1885. Jaccoud. Quoted from Letellier's Thesis, *q. v.*
1890. Model. Ueber Bronchitis Fibrinosa. Dissertation, Freiburg (4 cases).
1895. Magniaux. Thèse de Paris.
1896. Klein. Wiener klin. Wochenschrift.
1895. Sokolowski. Deutsch. Arch. f. klin. Med., lvi. p. 476.

GROUP VII. 5 Cases. *Cases showing a distinct relation to Asthma. Casts small.*

1864. Friedreich. Virchow's Archiv, Bd. xxx. S. 381.
1881. Pramberger. (See Group VI.)
1883. Vierordt. Berliner klin. Wochenschrift, No. 29.
1888. Wolf. Ueber Bronchitis Fibrinosa. Dissertation, Würzburg.

GROUP VIII. 4 Cases. *Fibrinous Exudation into the Bronchi associated with Pulmonary Œdema following Thoracentesis.*

1885. Scriba. Deutsch. Arch. f. klin. Med., Bd. xxxvi. (Cited in Ortner.)
1892. Hampeln. St. Petersburger med. Wochenschrift, S. 336. (Cited in Ortner).
1899. Ortner. Wiener klin. Wochenschrift, No. 44. (2 cases.)

GROUP IX. 6 Cases. *Cases unclassified through dearth of data.*

1873. Cohen. Transactions of Path. Soc. of Phila. (Laryngeal Tuberculosis.) Vol. v. p. 126.
1875. Siridez. In Championnière's Thesis, 1876.
1883. Escherich. Deutsch. med. Wochenschrift, No. viii.
1884. Möller. Second Case. Read in Schmidt's Jahrbuch., Bd. 204.
1888–89. Limont. Northumberland and Durham Med. Society.
1900. Lowenthal. Liverpool Medical and Chirurgical Journal.

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"Bronchite Pseudomembraneuse Aspergillaire Primitive."

1899. Devillers et Renon. La Presse Médicale.

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1895. Friedberger u. Fröhner. "Pathology and Therapeutics of Domestic Animals."

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1373. Peacock. Transactions of Path Soc. of London, vol. xxiv.
1880. West. British Medical Journal, vol. i. p. 282.
1893. Richards. Lancet, May, 1893.

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1864. Schnittzler. Wiener med. Halle, p. 450.
1868. Stoffeln. Oppolzer's Vorlesungen über Spec. Path. u. Ther., Bd. i.

SOME FOREIGN REFERENCES, NOT READ.

1896. Mills. Clinique Brux. "Apropos d'un cas de Bronchite Pseudomembraneuse. Recherches Bacteriologiques."
1896. Landrieux et Triboulet. Rev. gén. de Clin. et de Ther., vol. x. pp. 97–100. "Bronchite pseudomembraneuse aigne à pneumococques."
1897. Koren, F. Worsk. Mag. f Megeverdiensk, Kristiania, 4 R. xii.
1900. Prik. Riforma Med., Palermo, 1900, vol. i.
1900. Demel. Gros. d. r. Acad. di Med. di Torino.
1900. Vysiri. Casop lék ásk, Praha, vol. xxxix.

REVIEWS.

A Text-book of Physiologic Chemistry for Students of Medicine and Physicians. By Charles E. Simon, M.D., of Baltimore, Md. Philadelphia and New York: Lea Brothers & Co., 1901.

This text-book, while not so encyclopædic in scope as some of the standard German works on physiologic chemistry, contains all that the student of medicine and the physician for whom the book is written need know. The author has adopted the wise plan of avoiding all theoretic discussion, and limits himself to a clear, concise, and well ordered exposition of the facts of chemical physiology.

A careful review of the different chapters fails to reveal a single statement that is not fully in accord with the most recent research. The author is conservative, and does not often indulge in apodictic statements; where he does so, he is in full agreement with the best recognized authorities.

The book is fully up to date—the more recent discoveries in the field of the oxidizing tissue-ferments, of coagulation by nucleo-histon, of the purin bases, etc., are fully and adequately described and characterized. At the same time the classical facts of the science are presented in a precise and comprehensive manner. It also possesses two negative virtues. In the first place, the author wisely refrains from wandering into the clinical field; this temptation very few writers on this subject have been able to resist; the author has enhanced the value of his work by limiting himself strictly to the chemical and physiological aspects of the subject without attempting to teach pathological chemistry or chemical diagnosis; it is possible Dr. Simon realizes that he has treated these two subjects exhaustively in a separate publication, and is anxious to avoid repetitions.

In the second place, happily, all literature references are omitted. Whoever has been forced to seek his way through the protean array of names, titles, and page numbers that disfigure the average text-book of this character will heave a sigh of relief to find the path so clear and unencumbered. Literature references are of value to the original investigator and not to the student and the practitioner. It is really a matter of indifference to the beginner whether A or B or C has discovered this, that, or the other fact. Such is fame! Even the investigator, if he *is* an investigator, will not be content with those references that are given by any one author in a text-book; he will want the *complete* literature, and will consult an index of his specialty. Wherever we see too many names and too many quotations we place the burden of proof on the author to show that his book is not a mere compilation.

The book is a pioneer; it is the first large text-book of physiological chemistry that has been written in America; it is timely, for we are on the threshold of a new era in scientific medicine; physiological chem-

istry, we believe, is destined to throw much light into the obscure recesses that are around us and in front of us.

We welcome Dr. Simon's book most cordially; it fills a want and deserves a place on the shelves of every thoughtful and progressive physician. A. C. C.

A Laboratory Hand-Book of Urine Analysis and Physiological Chemistry. By G. Charles L. Wolf, B.A., M.D., Instructor of Physiological Chemistry, Cornell University Medical College, New York. Illustrated. Philadelphia: W. B. Saunders & Co., 1901.

This unpretentious little volume fulfils the purpose for which it is written. The first part of the book deals with some of the simpler exercises in physiological chemistry. In the second part, a brief sketch of the most common methods of urinary and gastric analysis are described.

Particular attention is given to clinical questions. There are a number of tables of urinary diagnosis at the end of the book that are conveniently arranged for reference, and present much that is described in the text in a diagrammatic form.

In the preface the author modestly disclaims completeness for his volume; the book certainly contains all that the average medical student at the average American medical college can assimilate within the short time usually allotted to the laboratory study of physiological chemistry—so that in the hands of a competent laboratory instructor the little book should prove a useful guide. A. C. C.

Irregularities of the Teeth and Their Treatment. By Eugene S. Talbot, M.D., D.D.S., Professor of Dental and Oral Surgery, Northwestern University Woman's Medical School, Chicago, etc. Fourth edition, with 580 illustrations. Pp. 537 and index. Philadelphia: The F. A. Davis Company, 1901.

A vast amount of research is represented in the work before us, much of it valuable and interesting, and much that is open to criticism, both as to its scientific accuracy and as to the deductions drawn. One gathers the impression from a critical study of the work that the author in his endeavor to defend his main contention of the constitutional origin of dental irregularities has belittled and practically ignored the evidence on the other side of the question, for unlimited and unanswerable data are at hand to demonstrate that many cases of dental irregularity do arise solely from local causes.

Many of the statements are involved and lack clearness, while others are self-contradictory; thus, at page 15 we find "the peculiarity in the size and shape of the jawbone may be inherited, but the manner of the eruption of the teeth is not transmitted, hence irregularities of the dental arch *per se* are not inherited. The muscles of the cheeks have nothing to do with the production of the V- or saddle-arch. The only

tissues involved are the jawbone on the one hand, the teeth and alveolar process on the other." The idea that irregularities of the dental arch *per se* could or could not be inherited presupposes a common understanding of the term "inherited." Teeth regular or irregular are not *per se* inherited in any strict sense, nor are any other organs for that matter. It is the developmental impress which is the physical heritage, a fact that the author fails to realize, or at least one which he ignores in many instances where the subject of heredity is being dealt with as an etiological factor. The dictum of the author with reference to the causal relation supposed to exist between mouth breathing and contracted dental arches cannot by any means be accepted as fitting all cases. It is with regard to this question especially, but by no means exclusively, that his statement is rather the contention of the advocate than the opinion of the judge. He asserts (page 16) "that the vault is not contracted by mouth breathing." "That mouth breathing (due to hypertrophy of the nasal bones and mucous membrane, deformities of the nasal bones, adenoids, or any pathological condition producing stenosis) does not cause contracted jaws, but all these conditions are due to neuroses of development. On the Continent of Europe the practical acceptance of these views has been shown in the recent translation by Max Bauchwitz of my work on the subject." This is jumping at a conclusion with a vengeance—that Max Bauchwitz saw fit to translate some of the writings of Talbot into German is scarcely evidence that the views of the latter have found practical acceptance on the Continent of Europe. On the contrary, there is on the Continent of Europe a wide spread belief among many thoughtful practitioners of medicine, surgery, and dentistry that mouth breathing due to nasal stenosis does cause a contraction or lateral flattening of the upper dental arch as well as faulty occlusion of the upper and lower arches.

Grevers, of Amsterdam, has shown very clearly the developmental part played by the tongue in giving form and proportion to the upper arch, and the further fact that the tongue cannot properly exert its moulding effect upon the arch when the mouth is continually kept open for breathing purposes.

Cryer (*Studies of the Facial Region*) has further shown the developmental effect of the percussive force of mandibular occlusion which is lost in the mouth-breather. These are factors which cannot be ignored, nor can they be included in Talbot's etiological classification as neuroses of development.

The author is not as precise in the use of his terms as one could wish, and, indeed, has the right to expect, in a really scientific exposition of any subject. His fondness for the theory of degeneracy has resulted in an undue importation into this work of matter related to degeneracy having only a remote and often questionable bearing upon irregularities of the teeth. Indeed, two thirds of the matter could have been omitted or condensed with benefit. In pursuit of his favorite theme of degeneracy he makes frequent allusions to "arrest of development" in explanation of certain defects. There are unquestionably many cases of arrest of development, but a retardation of development is not to be confounded therewith, which the author does in numerous instances.

In the preface the reader is directed to pursue numerous other works of the author in order to secure a comprehensive grasp of the whole subject, notwithstanding the fact that the author himself has exten-

sively drawn upon his other writings in the compilation of this one. Thirty-one of the thirty-two chapters are taken up with matters exclusive of the practical treatment of cases. The final chapter on treatment is an exhibit of the author's methods to the exclusion of all others, while throughout the work the use of the personal pronoun in the first person singular is rather oppressively apparent. Notwithstanding its flavor of egotism, dogmatism, intolerant critical attitude toward opposing views, many instances of bad English and misspelling of words, the book is one which anyone interested in irregularities of the teeth, their etiology and treatment, should read for the information it contains, but before it can be accepted as a standard authority on the subject it will need more thorough revision and correction. E. C. K.

ANATOMY, DESCRIPTIVE AND SURGICAL. By HENRY GRAY, F.R.S. Edited by T. PICKERING PICK, F.R.C.S., Consulting Surgeon to St. George's Hospital, etc., and ROBERT HOWDEN, M.A., M.B., C.M., Professor of Anatomy in the University of Durham, etc. A revised American, from the fifteenth English edition, with 780 illustrations, many of which are new. Philadelphia and New York: Lea Brothers & Co., 1901.

IN this new edition of *Gray* a marked change has been made from previous issues. It is not simply the reissuing of an old book with a few alterations and additions. A new editor, Robert Howden, has assisted the former editor, T. Pickering Pick, and the result is that the entire volume has been thoroughly revised, brought up to date, and its illustrations much improved.

This has been done without materially increasing its size, only eight pages having been added. This we believe to have been wise. As it now stands, complete in one volume, it still fills its place as the students' text-book as well as a satisfactory book of reference for the practitioner. The publishers state that 231 new engravings have been inserted. These are scattered through the book, many replacing old cuts. Owing to a rearrangement of material space has been saved in some places. Thus, the development of the peritoneum is now included in the section on embryology, and not in that on the organs of digestion. The surgical anatomy of the organs of digestion has been entirely rewritten and brought up more in accord with recent practice. There are a couple of most excellent new cuts, showing a posterior view of the kidneys, colon, and great vessels in relation to each other and to the bones. The cuts on the teeth *in situ* are decidedly superior to those in the previous editions. In the section on the Skeleton thirty-four additional cuts have been inserted besides those which replace old ones. The text in the chapter on Articulations has not been changed much, but many of the illustrations have been entirely redrawn. The muscles of the perineum are included under the section on Muscles, there being besides a separate chapter devoted to the surgical anatomy of the perineum. Instead of considering the heart in a separate section on the thorax, it is included in the section entitled the Blood and Vascular System. This section also describes the arteries and the veins.

The description of the lymphatic system bears evidence of careful revision; this applies not only to the purely anatomical part, but also

to that on its surgical application. This is of particular value in reference to the active interest which is now being taken in glandular infection in cancer and other diseases. The section on the Spinal Cord and Brain has been practically written over again and many of the illustrations redrawn.

The same careful revision has been carried through the chapters on the Cranial and Spinal Nerves.

The section on the Organs of Special Sense likewise bears evidence of having been carefully scrutinized and, where desirable, altered. Some new material has been added to the chapter on hernia. The section on General Anatomy and Embryology has been placed at the end of the volume instead of at the commencemont, as in the old edition. It has been entirely rewritten from a more modern stand-point. Instead of beginning with a description of the blood, it starts with the cell, its character and properties. Seventy-six additional illustrations have been inserted in this part of the volume, many being reproduced from the excellent atlas of His. In this section is included the development of the various organs, and taken altogether it forms a most valuable chapter both for study and reference. The previous voluminous index has been made still more complete by the addition of two pages. It makes the book particularly satisfactory in hunting up disputed points. There is no question that the original author of the work, Henry Gray, possessed in a remarkable degree the faculty of presenting anatomical facts in a clear, concise, and attractive manner, and it is a great pleasure and satisfaction to see that notwithstanding the numerous revisions to which the work has been subjected the genius of Gray is still preserved. More attention is given to the applied feature of anatomy both in regard to its topographical and surgical and medical aspects than was the case in previous editions. This, of course, is due to the advances made in anatomy in these directions in recent years, and makes the book more suitable for use at the present time than previous editions would be. In this edition half-tone illustrations have been used to a greater extent than formerly. The manner of illustrating text-books is a subject with which publishers are now grappling. What the ultimate outcome will be is hard to say. The combination of half-tone and colors as used in this work may be a solution of the problem. At all events a cut such as that of the popliteal artery, on page 580, in which the arteries are red, the veins blue, and the nerves yellow, leaves little to be desired. Concerning the press-work nothing need be said; it is faultless.

G. G. D.

A MANUAL OF MEDICINE. Edited by W. H. ALLCHIN, M.D. Vol. II. General Diseases (*continued*)—Diseases Caused by Parasites; Diseases Determined by Poisons Introduced into the Body; Primary Perversions of General Nutrition; Diseases of the Blood. Vol. III. Diseases of the Nervous System. The Macmillan Co., 1901.

As an additional reference work on Pathology and Practice the present collection of monographs by English authors may be confidently recommended. While it does not pretend to rival in completeness of detail the larger systems of medicine, it possesses the advantage of being

confined within limits which justify the title of a manual. The prevailing views on the etiology and pathology of the morbid conditions which at present are engaging the attention of investigators receive their due share of consideration. Thus, in the introductory chapter on Diseases of the Blood, a fairly comprehensive exposition of the actual state of hæmatology will be found, although the portion devoted to clinical laboratory work might with advantage have been more fully elaborated.

The third volume is devoted to Diseases of the Nervous System. It is enriched by numerous differential tables and an elaborate table of spinal localizations, besides numerous explanatory cuts and diagrams scattered through the text. Of practical value is the short article on the method of examining patients suffering with speech defects, especially for one who is not in the habit of seeing many nervous disorders, as is also the chapter on Focal Diagnosis, embracing only twenty pages.

In the article on cerebral vascular lesions the differential points between the various forms of hemorrhage are stated with apparent clearness if somewhat dogmatically, but one's faith in the author's accuracy is shaken by an unfortunate contradiction in the symptomatology. Thus, on page 70, it is stated that "ventricular hemorrhage is indicated by rigidity giving place to flaccidity of the limbs on both sides," while on the opposite page we read that in the same condition "there may coexist rigidity of the limbs on one or both sides." It is to be hoped that the opportunity to correct or explain such discrepancies will be afforded by the appearance of a new edition.

A cursory survey of the most common conditions usually seen by the general practitioner leaves one with the pleasant impression that the authors are not afflicted with the pessimistic views so often displayed by special writers on the subject of treatment. Where the disease is incurable some consideration is at least devoted to palliative measures. The freedom with which the bromides and, above all, the coal tar antipyretics are recommended in a great variety of conditions requiring sedative and analgesic medication is to be deprecated. The treatment advised in epilepsy follows the beaten tracks, no mention being made of the recent endeavors to control the disease by dietetic management, thus avoiding the danger of mental deterioration incident to bromide medication. Indeed, the author states that "mental deterioration takes place at least as rapidly in those who are treated by other drugs," thus apparently taking its ultimate occurrence for granted. In fact, throughout the work treatment other than by drugs receives too little attention. The fact that the experience and methods of English physicians are reflected in the book will not detract from its interest to American readers. The style of the work is uniformly excellent, and presents a pleasing variety born of its mixed parentage. R. M. G.

SURGICAL APPLIED ANATOMY. By FREDERICK TREVES, F.R.C.S. New Edition. Revised by the author, with the assistance of ARTHUR KEITH, M.D., F.R.C.S. In one 12mo. volume of 576 pages, with 80 engravings. Philadelphia and New York: Lea Brothers & Co.

Treves' *Applied Anatomy* has long been prized by medical students for its lucidity, brevity, and, withal, its comprehensiveness. We know of no book between whose covers is compressed so much valuable infor-

mation in such an attractive style. Interwoven with the dry structural details are rich similes, novel cases, many curiosities of surgery, and much reasoning as to why we are contextured thus. This does not build an anatomical memory for us, but it does establish a process whereby many facts are recollected with but little effort. But not only to the undergraduate has this unique little work proven of great value, but also to the graduate whose familiarity with anatomical data diminishes proportionately with his remoteness from the dissecting-room. Many details are absolutely useless to the practitioner; these are omitted, and only those points directly bearing on practice considered.

In the present edition much new matter has been incorporated; the old material has been thoroughly revised and in some portions entirely rewritten. Some of the old illustrations have been eliminated and several new ones added. We note with regret the absence of the relational anatomy of arteries with reference to their ligation. Beginning with the surface markings, a part is studied from without inward. The structures severed in the various operations, together with the dangerous neighbors, are pointed out. Fractures and dislocations are considered in comparatively great detail; indeed, in parts of the book anatomical descriptions are sacrificed to the discussion of surgery. In the text the references are few, and in those that are used, sometimes the journal alone, and often the journal and the year only, are mentioned. There are a few evidences of hurried composition in places, and numerous typographical errors.

Treves remarks the little trouble from hemorrhage attending brain operations, a phenomenon not noted in this country. In the same paragraph the measurements of the orbit are given in the English, and those of the eyeball in the French, system. The seton is still recommended for corneal ulceration. F. T. S.

Atlas and Epitome of Special Pathologic Histology. By Docent Dr. Hermann Dürck, Assistant in the Pathologic Institute; Prosector to the Municipal Hospital L. I. in Munich. Authorized translation from the German. Edited by Ludvig Hektoen, M.D., Professor of Pathology in Rush Medical College, Chicago. Liver; Urinary Organs; Sexual Organs; Nervous System; Skin; Muscles; Bones. With 123 colored illustrations and 60 lithographic plates. Philadelphia and London: W. B. Saunders & Co., 1901.

In this the second volume of his *Special Pathologic Histology*, Dürck completes his description of organic lesions, taking up the morbid histology of the liver, urinary organs, sexual organs, nervous system, skin, muscles, and bone. Like its companion volume of the well-known Saunders' *Medical Hand Atlas* series, it partakes more of the nature of an atlas, although the accompanying text is more than explanatory of the illustrations, containing, as it does, short descriptions of the normal histology of the organs, besides more detailed accounts of their abnormal macroscopic and microscopic appearance. The colored illustrations, of which there are 123, are of uniform excellence, and should be of great assistance not only to the student, but also to the trained pathologist as a matter of reference. For the sake of completeness we could wish that

some space and a few plates had been devoted to the neoplasms in their relation to the various organs considered, but this subject has been reserved for the third volume on general pathologic histology, only those tumors occurring solely in connection with definite organs (hypernephroma, adenomyoma uteri, etc.) being described in the present volume. The system diseases of the central nervous system and the diseases of the organs of special sense have also been omitted, but as the latter are usually the subject of separate text-books the loss does not mar the value of the work. H. H. C.

La Léprose. Par le Dr. Dom Sauton, Avec planches hors texte et 60 figures en noir et en coleurs. 8vo., pp. 496. Paris: C. Naud, Editeur, 1901.

The author of this elaborate monograph has devoted ten years to the special study of leprosy, and during that period has visited nearly every region of the earth in which cases of the disease are grouped. He is, evidently, thoroughly equipped for its study, whether in the laboratory or at the bedside. The evidences of close observation, keen reasoning, patient experimentation, and historic research are abundantly present in this comprehensive work. It is divided into fourteen chapters, any one of which might have been issued separately as a monograph, a statement which does not necessarily imply a lack of unity in the work as a whole. It is evident, therefore, that the reviewer must confine his attention to such portions of the book as, in his opinion, possess the greatest general interest. Even with such limitation there is an *embarras de richesses.*

The chapter on the History of the Disease might be read with interest and profit by every physician, whether specially interested in dermatology or not. The first traces of leprosy are shown to exist in Egyptian sculptures of the earliest Pharaonic dynasties, which represent the mutilations of its advanced stages. Moses, who, as our author well remarks, spoke the language of his day and had no intention of writing a treatise on pathology, confounded scabies, psoriasis, tinea, and syphilis under the head of leprosy. A somewhat similar confusion of these affections is found in the works of Hippocrates. The first genuine description of leprosy, under the name of elephantiasis, is found in Galen, *De re medica,* in the first century of our era. The disease was probably imported into America by the Spaniards. Be this as it may, there is documentary evidence of its existence in this country toward the end of the fifteenth century.

It is commonly taken for granted that leprosy was vastly more prevalent in Europe during the middle ages than at the present time, this opinion being largely based upon the numbers of mediæval asylums for lepers. Thus, for example, according to Mathieu Paris, there were 19,000 such asylums in Christendom in the year 1244, and of these, 2000 were in France. When, however, we recall the fact that the diagnosis of leprosy at the period referred to was faulty to such a degree that, in the opinion of Lancereaux, three-fourths of the cases of so-called leprosy were, in reality, cases of syphilis, it becomes apparent that this traditional opinion may well be questioned. Sauton goes much further than this, for he says that to-day "leprosy" would, perhaps, be more

common than it was in the thirteenth century if we included under it scabies and various other dermatoses and syphilis—a mistake which is still made in the extreme Orient.

In the course of this historical chapter Sauton makes a quotation from the *Philonium of Valescus de Tarenta*, and, in so doing, assigns the date of publication of this work to the year 1400. This manifest absurdity might be regarded as a misprint but for the fact that it occurs in two places.

In Chapter III., which treats of the geographical distribution of leprosy, the most interesting section, and the longest, is that relating to the Sandwich Islands, of which Molokai is selected for the sequestration of those affected with the disease. On this island there are two leper villages, called Kalawao and Kalaupapa, the former of which contains about 400 lepers and the latter 700. Kalawao is renowned as the site of the heroic self-sacrifice of Father Damien, whose work is continued by a number of men and women, most of whom belong to religious orders, and none of whom is named by Sauton except Father Wendelin. Notwithstanding the fact that this island is set apart for lepers, whose deportation thither is compulsory, there are, according to Sauton, scattered among the islands of the Hawaiian Archipelago as many lepers as reside in Molokai itself, *i. e.*, between 1000 and 1100. The dwellers in the leper colonies of Molokai might well adopt the celebrated line of Dante,[1] for hope, whether of recovery or return, they must abandon. Sauton tells us that he saw several persons at Molokai who for years had exhibited no symptoms of leprosy.

The chapter on Etiology is divided into four parts, of which the first is devoted to the discussion of the etiological influence of climatic and telluric conditions, alimentation, defective hygiene, sex, race, etc. The conclusion is that any cause, or set of causes, which impairs the resistance of the organism to disease in general renders it an easier prey to the bacillus of Hansen. Perhaps, also, the virulence of this organism is augmented by some of these causes, and especially by overcrowding and defective hygiene in general. An interesting observation, in this connection, is that certain dermatoses, such as scabies, eczema, and prurigo, may open the way to infection through the skin.

The second part of this chapter contains a discussion of the question of heredity in all its aspects. In this connection the reviewer cannot refrain from expressing the opinion that, from a practical stand-point, there is considerable hair-splitting in the definition of heredity. This statement refers particularly to the distinction that is drawn between "conceptional heredity," *i. e*, heredity through an infected ovum or spermatozoön, and "heredo-contagion," *i. e.*, infection acquired *in utero* after the ovum has undergone a certain degree of development. It seems manifestly impossible to determine whether a child born with the signs of leprosy, or exhibiting them shortly after birth, has been developed from an infected ovum or has been infected *in utero*. It is established that the bacillus of Hansen may be present in the seminal fluid and in the ovum, but it is a mere matter of opinion whether or not an infected ovum is capable of development. Sauton holds the view that conceptional heredity or true heredity, as he calls it, involves sterility, abortion, or non-viability. To the reviewer it seems an ultra-refinement of terms, if not an absurdity, to designate as true heredity ("l'hérédité vraie") a condition which prevents inheritance. The third and fourth

[1] "Lasciate ogni speranza voi ch'entrate."

parts of this chapter deal with the subject of contagion, and in the latter there is a detailed account of fifty-three examples of the communication of the disease.

The chapter on bacteriology occupies seventy pages, and is profusely and beautifully illustrated. The physical properties of the bacillus of Hansen and its color reactions are described in detail. The question whether it can be cultivated is left undecided until the statements of Carasquilla are more thoroughly investigated. This observer, in a communication to the National Academy of Medicine of Bogota, claims success in his attempts at cultivating the organism, and his methods are detailed by Sauton. Whatever different opinions may exist concerning the culture experiments, all authorities seem to agree in the belief that all attempts to inoculate animals, including man, with leprosy have failed. It is true that a Hawaiian criminal who was inoculated by Arning developed leprosy some two years later, but the experiment is inconclusive, inasmuch as the man belonged to a leprous family and lived in an infected neighborhood. Certain points of resemblance between the bacillus of Hansen and the bacillus tuberculosis, as well as the prompt and decided reaction of lepers to the hypodermic injection of tuberculin, conduct Sauton to the conclusion that these two organisms have probably issued from a common stock. The most remarkable fact concerning the bacteriology of leprosy is the occasional disappearance of the bacillus of Hansen from tissues and organs in which it had previously abounded. It is now established by the authority of the International Congress of Berlin of 1897 that the diagnosis of leprosy must occasionally rest solely upon clinical evidence.

Sauton regards the customary clinical division of leprosy into tubercular and anæsthetic varieties as convenient for description, but artificial. It is too diagrammatic. Nevertheless, it is impossible for the writer to pursue his way among the labyrinth of symptoms or to act as a guide to others without some such clew. Our author, in common with other authorities, finds a schematic division indispensable, and adopts the following: The first period is that of infection, characterized by the primary accidents or symptoms. These consist of fever, progressive anæmia, headache and vertigo, digestive disorders, coryza, and epistaxis, and various sensory disorders. Among these various symptoms the only one upon which a diagnosis may be based is rhinitis. In the nasal discharge the bacillus of Hansen may frequently be detected.

The secondary period comprises the multiform tegumentary and mucous lesions, and also sensory disorders, among which the well-known "dissociation symptom," once supposed to be pathognomonic of syringomyelia, may be present.

The tertiary period is characterized by the predominance of trophoneurotic lesions, which may manifest themselves in the skin, the muscles, and the bones. The extraordinary resemblance of leprosy to certain diseases lately described under the names of syringomyelia, Morvan's disease, ainhum, Raynaud's disease, etc., is referred to in Chapter VII., in which the statement is made that the majority of cases exhibited as types of syringomyelia and Morvan's disease have, on closer study, been proved to be cases of leprosy. It is in great part owing to the work of Zambaco Pacha that the leprous character of many of these cases has been recognized.

The foregoing remarks suffice to indicate the nature and scope of this

monograph, which should be in all our medical libraries. It remains to add that the principal object of Dr. Sauton's labors was not to write a classical treatise upon leprosy, but to found in France a sanatorium for lepers. With this end in view he has purchased a tract of land near Neufchateau (Vosges), upon which, as he tells us, his charitable design would have been executed but for a veritable insurrection among the inhabitants of the neighborhood.

The work is dedicated to the memory of Pasteur, "cet illustre pionnier de la science et de la charité." F. P. H.

THE READY-REFERENCE HANDBOOK OF SKIN DISEASES. By GEORGE THOMAS JACKSON, M.D., Chief of Clinic and Instructor in Dermatology, College of Physicians and Surgeons, New York. New (4th) edition, thoroughly revised. In one 12mo. volume of 617 pages, with 82 engravings and 3 colored plates. Philadelphia and New York: Lea Brothers & Co., 1901.

THE thorough revision to which Dr. Jackson's well-known *Handbook of Skin Diseases* has been subjected brings it fully up to date. The more important recent additions to our knowledge of cutaneous diseases are appropriately mentioned, and the new remedies and methods of treatment are succinctly described. Considerable new matter has been added in the shape of sections on Acne Keratosa, Acne Urticata, Caraté, Crawcraw, Endothelioma, Erythrodermie Pityriasique en Plaques Disseminées, Fordyce's Disease of the Lips, Granuloma Necrotica, Impetigo of Bockhardt, Lichen Annularis, Lichen Pilaris, Pityriasis Lichenoides Chronica, and Verruga Peruana. These maladies belong among the less common dermatoses, except Fordyce's Disease of the Lips, which occurs in such a large proportion of persons, if it is not present in some degree in all, that it is rather doubtful if it should be regarded as a diseased condition at all.

The successful application of light and the Röntgen ray to the treatment of lupus vulgaris is briefly mentioned, but the very remarkable results reported in the past year or two following the use of the same measures in epithelioma of the skin are, through some oversight, not even referred to.

The section on Psorospermosis Follicularis Cutis might very well have been entirely omitted and the space given to some more important subject, since it is now admitted that the affection thus named is not a psorospermosis at all, but belongs to the keratoses—that so far as we know there is no such thing as a cutaneous psorospermosis.

We regret that some mention has not been made of the deep suppurating forms of trichophytosis which follow infection from the domestic animals. A considerable number of cases of ringworm, in which the deeper parts of the skin were invaded by the trichophyton, causing more or less extensive suppuration, are on record in the dermatological literature of the past few years, and it is time that some account of this important variety of the disease found its way into the text-books.

Of the new illustrations special mention should be made of the beautiful plates representing the eruption of variola; these leave little to be desired in the way of pictorial illustration.

Upon the whole, this new edition of Dr. Jackson's book well maintains the reputation acquired by its predecessors as a reliable guide in dermatology. M. B. H.

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MEDICINE.

UNDER THE CHARGE OF

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Observations on Carcinoma of the Stomach.—Boas (*Arch. f. Verdauungskrankheiten*, 1901, vii., 413) communicates a number of interesting observations made on 141 cases of carcinoma of the stomach occurring within the last five years. He treats especially of the manner of onset, course, and complications of the disease, as well as of the condition of the intestine, together with some observations concerning the urine.

In thirty-nine, or more than a quarter of all his cases, the symptoms began abruptly. In the words of the author:

"1. The onset of carcinoma of the stomach is by no means always gradual; it is often wholly acute, with or without slight prodromata scarcely recognizable to the patient or the physician.

"2. Sometimes the onset manifests itself by a single sudden hemorrhage from the mouth or anus occurring during apparently perfect health."

If several hemorrhages follow in rapid succession the differential diagnosis between simple ulcer and ulcus carcinomatosum or carcinoma may be very difficult. If the patients are of advanced age one should, under all circumstances, think of carcinoma. Boas observes that about one-third of the cases retain their appetite, or even show an increased desire for food. The condition of the appetite in carcinoma of the stomach may be most variable. With regard to the tongue, the author states: "According to my experience, there can be no question of any recognizable connection between the serious pathological process in the stomach and the tongue." Not infrequently the tongue may be perfectly clean. The decided majority of his cases showed constipation.

Enlargement of the supraclavicular glands on the left side is an extremely rare symptom. It was not present in a single one of the 141 cases. Three instances of secondary carcinoma of the umbilicus are reported. While this

is, as a rule, a late symptom, it may occur early in the course of the disease, so as to be the first diagnostic sign.

Albuminuria is a rare symptom; it occurred in but three of Boas' cases. In only one instance was glycosuria found. In five cases there was a complicating pulmonary tuberculosis. The diagnosis of ulcus carcinomatosum is often impossible. If the history points to a previous gastric ulcer, one would naturally suspect its relation to the existing malady, but the proof can only be obtained by autopsy.

Diagnostic Value of Blood Examinations in Gastric Ulcer and Carcinoma.—RENCKI (*Arch. f. Verdauungskrankheiten*, 1901, vii., pp. 234, 392) studied the blood in fifteen cases of carcinoma and fourteen of ulcer of the stomach. The red and white corpuscles were counted, the hæmoglobin estimated, and differential counts made of the leucocytes. No characteristic differences could be made out between the anæmia in carcinoma and ulcer, and the conclusion was reached that the examination of the blood is of relatively little value from a diagnostic point of view.

As a result of further studies in normal and various pathological conditions, in eleven cases of carcinoma ventriculi and in ten of gastric ulcer and benignant pyloric stenosis, the author arrives at the following conclusions:

1. During the digestion of albumin there appears, in the majority of cases, an increase in the number of white blood-corpuscles—a digestive leucocytosis.

2. The climax occurs generally in the third and fourth hours, the average increase in the colorless corpuscles amounting to 3543.

3. Its appearance is connected with the normal function of the pylorus and the intestine.

4. The absence of the digestive leucocytosis is dependent upon pathological conditions of the digestive tract.

5. The presence of an increased number of white corpuscles during digestion is of no value as a diagnostic symptom in cases where there is a question of doubt between new-growth and gastric ulcer, for it may appear in both conditions and is independent of the nature of the disease.

6. The absence of digestive leucocytosis in healthy individuals may depend upon a diminution in the contractile power of the pylorus, which results in its insufficiency.

The Westphal-Piltz Pupil Phenomenon.—SCHANZ (*Berlin. klin. Wochenschrift*, 1901, xxvii., 1065) states that Westphal, in 1899, called attention to a phenomenon present only in cases where the pupillary reflexes were abolished, with moderately or widely dilated pupils, and also in instances where the light reflex was absent, with retention of the convergence reaction. This phenomenon, which he failed to find in normal pupils except in one instance of hysteria, consisted in the fact that when the patient contracts the orbicularis muscle forcibly while the eyelids are held open, the pupil becomes appreciably contracted, while the eyeball turns upward and outward. Westphal believes that in forcible closing of the eyelids the pupillary branch of the oculomotorius and the sphincter iris muscle yet come into play even in pupils where the light reflex is abolished. The contraction of the pupil

is then an associated movement, and results from the same mechanism as that determining the upward and outward rotation of the eye. At about the same time Piltz made a similar observation, and called attention to the fact that the symptom, while present in a large number of patients with progressive paralysis, tabes, catatonia, epilepsy, dementia præcox, etc., was considerably commoner in progressive paralysis than in tabes, so much so as to be of some value in diagnosis. Galassi, who has observed this symptom previously, believed that in incomplete peripheral paralysis of the oculomotorius, with loss of all pupillary movements, this reaction might yet be brought about by energetic closure of the lids. The symptoms might, he thought, be of value in distinguishing a peripheral from a central paralysis. Antal, in 1900, studied this phenomenon and came to the conclusion that it was present as a prodromal symptom when the light reflex was still retained, as well as a late symptom in pupils with absence of light, accommodation, and convergence reflexes. Parisatti, however, in 1887, called attention to the fact that the mere increase of pressure in an eye might cause this symptom. Schanz has paid especial attention to the phenomenon, and appears to have accumulated proof of this latter view. The phenomenon was clearly demonstrable in one case after the use of atropine and in another instance of complete ophthalmoplegia interna of long duration.

In view of these cases the author believes that the possible action of the paralyzed nerve branch is out of the question, and concludes that the phenomenon is purely mechanical, due to an engorgement of the vessels of the iris from pressure. This phenomenon is probably masked in the normal eye by the light reflex. It is natural, therefore, that it should be observed only in pupils which are not wholly normal, at best when moderately dilated. In the absence of light reflex it may be missed owing to the narrowness of the pupil; this may well account for the fact that Piltz found the symptom less frequently in tabetics than in paralytics. The completeness of the reaction may depend upon various movements: the power of the orbicularis, the position of the eyeball, the hardness of the sclera, etc. The author has observed the phenomenon, also, in cocainized eyes. He concludes: "According to my opinion, this phenomenon is purely mechanical, and the only conclusion which can be drawn from it is that the innervation of the pupil is not wholly normal, for otherwise the light reflex would hide this mechanical narrowing. All further conclusions which rest upon the idea that it is an associated movement similar to the rolling upward of the eyeball on closing of the eyelids appear to me erroneous."

Primary Cancer of the Bile-ducts.—DEVIC and GALLAVARDIN (*Revue de Médicine*, July, August, and October, 1901, pp. 557, 658, and 839) report a case of primary carcinoma of the common bile-duct and give an exhaustive analysis of a total of 55 cases of primary cancer involving various portions of the bile passages, which they have collected from the literature. Primary cancer of the gall-bladder and of the ampulla of Vater are not included. Only those cases in which the hepatic duct and its branches, the cystic duct and the common duct (exclusive of the ampulla) are involved are included in the series.

From the fact that they have been able to find only 55 cases, they conclude

that the condition is rare. In 46 cases in which the sex was mentioned, there were 30 males and 16 females. This is entirely contrary to what is the case in cancer of the gall-bladder, where from 75 to 80 per cent. are in women. Gallstones are less frequently mentioned as a probable cause than in cancer of the gall-bladder. The largest number of cases occur between fifty and sixty-six years of age. In 53 of the cases the distribution of the growth was as follows: The common duct was involved in 22; the junction of the hepatic, cystic, and common ducts in 15; and the hepatic duct and its branches in 16. In about one-fifth of the cases metastases occurred, these being practically confined to the liver.

The condition of the gall-bladder as regards dilatation was of interest. Of 18 of the cases of cancer of the common duct in which reference to the gall-bladder was made, it was dilated in 17 and normal in 1; of 14 of the cases where the growth was at the junction of the common, hepatic, and cystic ducts, the gall-bladder was dilated in 7, normal in 3, and contracted in 4. In only 7 of the 16 cases of cancer of the hepatic duct and its branches was reference made to the condition of the gall-bladder. It was dilated in 2, normal in 1, and contracted in 4.

In the majority of the cases the liver was enlarged. Of 37 cases in which its condition was noted it was hypertrophied in 25, and either atrophied or normal in 12. Primary cancer of the bile-ducts is characterized by a chronic jaundice which is insidious in its onset and of gradually increasing intensity. Digestive disturbances occur. Pain is not a prominent symptom. Progressive emaciation and a decided cachexia precede death, which occurs usually in from two to six months. The examination of the abdomen practically never reveals any tumor. The spleen may be increased in volume. Ascites occurs in about half of the cases.

The writers divide the cases clinically into four groups, as follows:

1. The usual type, with hypertrophied liver and enlarged gall-bladder, comprising almost all of the cases of cancer of the common duct, and some of the cases with involvement of the hepatic duct.

2. The atypical form with or without hypertrophy of the liver, and atrophy of the gall-bladder, found chiefly with cancer of the hepatic duct.

3. A form simulating hypertrophic biliary cirrhosis, owing to the enlargement of the liver and spleen.

4. A form without jaundice. This is extremely rare, only one case having been reported.

Diagnosis is not easy and it has to be differentiated from hypertrophic biliary cirrhosis, obstruction of the common duct by a calculus, and from obstruction of the common duct by an adjacent cancerous gland or a cancer of the ampulla of Vater or cancer in the head of the pancreas.

The treatment is chiefly surgical, and then merely palliative in the form of a cholecystostomy or cholecystenterostomy.

Cyto-diagnosis and Tuberculous Meningitis. — MARCOU-MUTZNER (*Archives Générales de Médecine*, September, 1901, p. 345) reports a case of tuberculous meningitis, in which he studied the character of the cells in the cerebro-spinal fluid, the result leading him to cast doubt on the value of "cyto-diagnosis" in the differentiation of various forms of meningitis.

Widal, Sicard, and Ravaut were the first to draw attention to the possibility of making a diagnosis of the form of meningitis from the morphology of the cells contained in the cerebro-spinal fluid. Subsequently it was believed possible to determine the cause of the development of pleural, peritoneal, and joint fluids from the character of the prevailing cell present in these exudates.

Milian has recently formulated the results obtained by the study of the cells in the various fluids as follows:

1. If the prevailing cell in the cerebro-spinal, joint, or hydrocele fluids be the mononuclear lymphocyte, the inflammatory process is tuberculous in origin.

2. If, on the other hand, the polymorphonuclear leucocyte be the prevailing cell, the condition is due to some other organism than the tubercle bacillus.

3. In the case of pleural and peritoneal exudates, as well as of hydrocele fluid, if the predominant cell be an endothelial cell, the fluid is the result of some mechanical disturbance, such as cardiac or renal disease.

Marcou-Mutzner's case was a man forty years of age, who had been gradually losing weight for several months. For two months previous to admission to the Charité Hospital, he had suffered from a right-sided sciatica, for which he sought treatment. There were signs of local consolidation at the apex of the right lung. During the patient's stay in the hospital he developed definite signs of meningitis, including Kernig's sign. Lumbar puncture was performed, and the cerebro-spinal fluid, which was fairly clear, was centrifugalized, and the sediment stained and examined microscopically. The prevailing cell was found to be the polymorphonuclear leucocyte. This occasioned surprise, as the case had previously been regarded as one of tuberculous meningitis. As a result of the examination of the spinal fluid, however, the diagnosis was changed to that of acute meningitis of non-tuberculous origin.

The patient died three days later, and an autopsy was performed. He was found to have typical pulmonary and meningeal tuberculosis.

The writer cites another case reported by Rendu, in which the patient was admitted to the hospital comatose and aphasic. The examination of the cerebro-spinal fluid showed that the lymphocyte predominated. A diagnosis of tuberculous meningitis was made. The autopsy eventually showed a fracture of the base of the skull.

These cases cause Marcou-Mutzner to doubt the value of the study of the cells in the cerebro-spinal fluid in meningitis, and he concludes that it is possible to have an excess of the polymorphonuclear leucocytes in the spinal fluid in tuberculous meningitis, and also to have an excess of the lymphocytes without the latter disease being present.

Results Obtained by Antityphoid Inoculation in the Case of an Epidemic of Typhoid Fever which Occurred in the Richmond Asylum, Dublin.—WRIGHT (*The Lancet*, October 26, 1901, p. 1107) gives the results obtained from the inoculation with antityphoid vaccine of the inmates of the Richmond Asylum during the prevalence of an epidemic of typhoid fever in that institution. The first case of the epidemic came under obser-

vation on August 7, 1900, and from that date on to the end of December, 54 cases of typhoid fever occurred among the patients and the nursing staff. No reference is made to the probable source of the infection. Wright was applied to for a supply of antityphoid vaccine. The first batch of 45 inmates was inoculated on September 6th, 0.75 cubic centimetre of vaccine being used at each inoculation. By November 30th, after a series of seventeen sittings, a total of 511 persons had been inoculated. Excluding all patients above fifty-five years of age as insusceptible, Mr. Cullinan, who made the inoculations, estimated that the total number of susceptible persons exposed to infection on the date on which the inoculations were begun was 655. Of these, 541 were insane inmates, the balance of 114 being nurses. Of the 541 inmates, 511 were ultimately inoculated. None of the 114 nurses received the protective serum. According to Mr. Cullinan's report, 29 and 7 cases of typhoid fever, respectively, occurred in the uninoculated and inoculated groups after the commencement of the treatment on September 6th. Wright claims that 2 of the 7 cases must have contracted the infection before the inoculations were made, thus leaving only 5 cases among the inoculated. He does not consider that the uninoculated nurses were more exposed to infection than the inoculated inmates. A further comparison is made by considering the number of cases that developed in the two groups after the completion of the inoculations. Thus in 114 uninoculated persons 5 cases developed, whereas in 504 inoculated inmates (excluding the outside limit of 7 cases that previously developed the disease) no cases occurred. Wright thinks that these statistics prove the undoubted protective power of the vaccine, and says they agree well with the results which have reached him from other sources.

On the Presence of Micro-organisms in the Circulation during Pneumonia.—PROCHASKA (*Deutsch. Arch. f. klin. Med.*, Band lxx. p. 559), continuing his early observations in this field (*Centralbl. f. Gen. Med.*, 1900, No. 46)—in the clinic of Prof. Eichhorst—has examined bacteriologically the blood of forty consecutive cases of pneumonia. The blood, nearly 10 c.c. in amount, was collected by a sterilized syringe from one of the arm veins after elastic constriction and thorough cleaning of the skin. Control experiments with normal individuals proved the reliability of this method; in no instance were contaminations obtained.

Forty cases were examined, of which seven were fatal. In thirty-eight instances the pneumococcus was obtained. Twice there developed organisms, concerning the nature of which the author is uncertain. These were streptococci, with a tendency to arrangement in pairs; they may have been an especially virulent variety of pneumococcus. One of these cases ended fatally; in the second slow resolution was followed by a secondary indurative process.

The cultures were made at varying periods of the disease, sometimes as early as the second day. In two cases positive results were obtained on the day following the crisis; in one of these there developed later a muscular abscess. In one case, followed by an empyema, pneumococci were found in the circulation on the second day after the crisis. In another instance with slow resolution, organisms were found three days after the febrile crisis.

Prochaska's observations are not sufficient to justify comment on the suggestion of Paesller that the number of organisms in the circulation and the degree of leucocytosis may bear an inverse relation one to another.

The author believes that his positive results are due in part to the large quantities of blood used, and in part to the use of bouillon as a culture medium.

Adding to these cases those mentioned in his previous publication, Prochaska is able to report fifty cases of pneumonia in which septicæmia has been regularly demonstrated; and only twelve of these cases resulted fatally. The number of organisms in the circulation varied greatly in different instances, having apparently no relation to the severity of the case. These results appear to justify the conclusion that pneumococci are always present in the blood during pneumonia. The demonstration of this fact affords a ready explanation of the frequent metastatic complications of pneumonia due to diplococcus lanceolatus, occurring, as they not infrequently do, in mild as well as in severe cases.

SURGERY.

UNDER THE CHARGE OF

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Three Cases of Pulmonary Abscess, with Operation.—SMITH (*Yale Medical Journal*, February, 1901) states that the indications for the operation of pneumotomy are: tuberculous cavities in exceptional cases, where the disease is confined to the lung; bronchiectasis, hydatid cyst, gangrene, abscess, foreign bodies, and hemorrhage. The results of operations for tubercular and bronchiectatic cavities are not especially encouraging. With the former, tubercular disease is left after the operation has been performed and new cavities will probably occur. With the latter, temporary relief or improvement is all that can usually be expected, for there will be other smaller cavities that will burrow and enlarge. Hydatid cyst of the lung is best treated by pulmonary incision, and the results are very encouraging. Reclus reports eleven operations with but two deaths. Gangrene should be treated by incision, if the disease is not diffused and yet is causing symptoms of septicæmia. Statistics prove that over 50 per cent. of these hitherto fatal cases can be saved by early and efficient operation. Abscess of the lung, which is not being drained through the bronchi, or if being drained is wearing the patient out by prolonged fits of coughing, or where the evidences of septic absorption are pronounced, should be opened and thoroughly drained, unless some strong contraindication exists. Injuries to and foreign bodies

in the lung may call for operation, especially when inflammatory action and the formation of abscess have occurred. Hemorrhage of the lung from traumatism, which cannot be controlled by ordinary methods, may possibly be checked by incision and ligation—an operation that has been but rarely done. Gangrene is diagnosed by the history, the physical signs, severe constitutional symptoms, together with the fetid breath and characteristic sputum, which is greenish-brown and contains elastic fibres arranged in an alveolar manner, and having an unequalled odor. The diagnosis of abscess of the lung is usually not difficult. There are the physical signs of dulness, increased vocal resonance and fremitus, and frequently the sound of air passing through a cavity filled with fluid. The sputum is very offensive and usually of a brownish color. The microscope shows septic micrococci, bits of lung tissue, with crystals of fat and hæmatoidin. In acute cases the constitutional symptoms are usually severe, septic absorption commencing early. This is probably accounted for by the inefficient walling off of abscesses in the lung tissue. The exploring needle will sometimes find pus, but may fail even in cases where it is present. It may serve the purpose of establishing whether the pleural surfaces are adherent, for if so the needle will oscillate but slightly when introduced, but if not adherent it will oscillate synchronously with respiration. Among the dangers and contraindications of the operation are that there must always be some danger from an anæsthetic given at a time when the patient is seriously ill. Chloroform is probably the best anæsthetic in most cases, as it provokes less irritation of the respiratory organs than ether. Many surgeons advise against operation when the pleural surfaces are found free, but the author believes that there is better hope of success in operating at once than in waiting for adhesions to follow a primary operation, for in no one of the author's cases was the patient likely to have survived unaided that length of time. Septic infection in all cases must be scrupulously guarded against. There is but little danger from hemorrhage, and if there be any it can usually be easily controlled. The best incision is one following the curve of the ribs from 6 to 12 cm. in length over the seat of the disease. This incision should be made over the centre of a rib, if one is to be resected; if not, the opening is to be made in the intercostal space midway between the ribs. The intercostal muscles are divided and the costal pleura examined. If a rib is resected, from 3 to 4 cm. should be removed by the curved bone forceps or the Gigle saw, after separating the periosteum by means of a periosteal elevator. It should next be determined if the pleural surfaces are adherent. The edges of the wound are held apart by retractors and an aspirating needle or trocar and canula should be introduced. If pus is found and the pleura are adherent a blunt instrument or the finger should be carried into the lung in the track of the aspirating needle. If they are not adherent one must decide between the dangers of an immediate operation and possible infection of the pleural cavity, and the dangers of postponing further operative interference until adhesions are formed or the abscess ruptures. The author has had good results with irrigation in those cases where there was an offensive discharge. If the pus is not offensive and there is good drainage irrigations are not necessary. The future of this operation should be one of progress. There should be more thorough understanding of its applications, its limita-

tions, and a readiness on the part of practitioners to give their patients the benefit of it when necessity arises.

The Surgery of the Spleen.—WARREN (*Annals of Surgery*, May, 1901) states that splenectomy is no new operation, and that the number of operations performed in modern times is by no means small. Hagen has collected 360 cases, with a mortality of 38.3 per cent. After carefully revising his list and eliminating what would be considered incurable cases and cases in which the diagnosis has not been sufficiently established, Hagen was able to reduce this mortality to 12.2 per cent. (1) Malarial spleen. Hagen has collected 88 cases of malarial hypertrophy of the spleen, exclusive of wandering spleen. Of these cases, 24 previous to the year 1890 gave a mortality of 62.5 per cent., while 64 cases operated after the year 1890 gave a mortality of 23.4 per cent. When one considers the very large size that the organ often attains in this disease, and the unfavorable constitutional condition of the patient, such results are at least encouraging. (2) Splenic anæmia or splenic pseudoleukæmia. Sippy reports 7 cases in which splenectomy was performed, with 5 recoveries. Osler has reported 15 cases, 1 of which was operated upon, with a fatal result. Warren reports 1 successful case. (3) Splenic leukæmia. Splenectomy in this disease is almost invariably followed by a fatal result. Hagen reports 42 cases, with only 4 recoveries. Death was almost, without exception, due to secondary hemorrhage from the surface of the wound, owing to the condition of the walls of the large vessels. Richardson has had one successful case. (4) Chronic enlargement of the spleen in infancy is generally secondary to some other disease, and yields rapidly to treatment appropriate to the causal condition. (5) Banti's disease, or hypertrophy, with cirrhosis of the liver. Hagen reports 16 cases in which splenectomy was performed, with only 3 deaths. (6) Wandering spleen. The number of cases operated upon up to the last decade has been comparatively few, but during this decade 11 reported cases, with 4 deaths, giving a mortality of 36.3 per cent. On the other hand, of 43 cases of wandering spleen operated upon during the last decade there were only 3 deaths. It would seem, therefore, that as a prophylactic measure extirpation of the wandering spleen was a justifiable procedure, and that we should certainly be within the limits of propriety in advising an operation where the displacement had reached a degree to produce marked abdominal symptoms. Splenoplexy does not seem to meet with approval, as the result is uncertain. (7) Abscess of the spleen. Only certain forms of abscess of the spleen are suitable for splenectomy. Such cases are those in which the spleen itself is surrounded with pus, or cases in which the spleen, containing an abscess, is not too tightly bound down to the abdominal wall by adhesions. Where there is danger of infecting the general peritoneal cavity by an attempt to extirpate the spleen it would be better to simply open the abscess and drain. Hagen has collected 7 cases of abscess, all of which recovered after splenectomy. In 1 of these cases the abscess followed appendicitis. The same author has also reported 3 cases of splenectomy for tuberculosis of the spleen, with 2 recoveries. (8) Rupture of the spleen. Pitts reports 3 successful cases of splenectomy for rupture. Sevor reports a successful case. Since 1890, 34 cases of splenectomy for rupture of the spleen have been reported, with a mortality of 41.2 per

cent. (9) Sarcoma of the spleen. Hagen, up to the year 1890, has reported 5 cases of splenectomy for sarcoma, of which 2 died and the remaining 3 recovered. From 1891 to 1900, 4 cases have been reported, with only 1 death. After reviewing at length the technique and sequelæ of splenectomy Warren concludes with the report of 5 cases: (1) Splenic anæmia, splenectomy; recovery. (2) Sarcoma of the spleen, splenectomy; death. (3) Splenic leukæmia, splenectomy; recovery. (4) Rupture of an infected spleen, splenectomy; death. (5) Splenopexy, recovery.

Circumcision as a Preventive of Syphilis and Other Disorders.—FREELAND (*Lancet*, London, December 29, 1900) states that if it were possible to secure the efficient circumcision of every male in infancy not only would many of the disorders incidental to the genito-urinary organs in childhood, adolescence, and adult life be prevented, but also the frequency of syphilis would be materially diminished. It is necessary to adduce good evidence, not only that the results justify the operation, but also that the operation is justifiable. Therefore, it is necessary to show that the operation is free from risk, that the deprivation of the structure which it is proposed to remove will not inflict any physical disability on the individual, and that the benefits which are likely to accrue are substantial and commensurate with the sacrifice. Everyone has agreed that the operation of circumcision in a healthy child by a competent surgeon is attended with such infinitesimal risk that for all practical purposes it may be considered safe, and that from a sanitary point of view the removal of the prepuce is a decided advantage and does not in any way interfere with the physical welfare of the individual, but instead is a distinctive advantage. It is the prevalent opinion that syphilis is less common among the Jews, but actual statistics are few. Hutchinson analyzed 330 cases of venereal diseases among hospital patients, 58 of whom were Jews, 47 of these had gonorrhœa and 11 had syphilis. The author states that his own experience completely bears out the idea that syphilis is much less prevalent among those who are circumcised. Statistics collected by Fournier, Clerc, and Hill show that in 898 cases of initial lesion 659, or 73.3 per cent., appeared on the prepuce or just behind the corona. Probably the veriest enthusiast would hardly claim that the whole of this 73.3 per cent. of chancres could be prevented by circumcision. One may conclude, however, that the universal practice of circumcision should reduce the case incidence of genital syphilis in the male by something between 25 per cent. and 73 per cent., or, taking the mean of these, by 49 per cent. In syphilis it is not only the guilty who suffer, but the innocent suffer with the guilty, and the sins of the fathers are in very truth visited upon the children. There is direct evidence to show that the practice of circumcision will materially diminish this massacre of the innocent. Hutchinson found that out of 97 female hospital patients that came under his observation in one year for venereal disease, 92 were Christians and 5 were Jews; of the former 6 were married, and at least two-thirds of these had apparently contracted syphilis from their husbands. The same observer found that out of 252 children under five years of age who came under his care in the course of a year there were 179 Christians giving 27 cases of congenital syphilis, and 73 Jews giving only 3 cases of congenital syphilis.

These statistics clearly show an enormous reduction in the case incidence of syphilis among the women and children of the circumcised Jews, and in view of what has already been said the only deduction which can reasonably be drawn from their study is that if the practice of circumcision were to become as general among Christians as it is among Jews a proportionately great reduction of the case incidence of syphilis would take place among their women and children, and this amply proves that the end justifies the means.

PEDIATRICS.

UNDER THE CHARGE OF

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Intra-uterine Rickets.—F. C. ABBOTT, of London (*British Medical Journal*, September 7, 1901, p. 597), describes under this title the physical appearance and post-mortem findings in a female child first seen by him a few weeks before death, at the age of fifteen months. The child was one of twins, the last children of healthy parents. The mother altogether had had seven living children, as well as eight miscarriages; these included three pairs of twins, one of each of the two previous pairs had, however, been a "false conception" and born dead. The living children were all well. The last pair of twins, one of whom was the patient, were, in the mother's opinion, three weeks premature. She was well when carrying them, and knew of nothing abnormal in her diet during the pregnancy. The mother was certain that the baby's legs were at birth quite the same as at the time of observation.

At the time of death the child showed marked rhachitic changes in the trunk and abnormal curvatures of the limbs. Many of these curves were the ones commonly associated with rickets and usually explained as being due to muscular tractions, or to methods of progression and support of the body weight. It is interesting and suggestive, however, in the author's opinion, that many of these curves could be produced entirely by intra-uterine pressure, such as the longitudinal chest groove for the arms to rest in, and the curve of the femora against the trunk. This latter was exactly the same curve as was noted in Makin's case (a baby fourteen days old). Some of the curves, on the other hand, were quite unusual, and could only be explained by the intra-uterine folding of the child. These were the curve of the lower end of the forearm with its convexity forward and inward, and the curve of the right leg, with its convexity forward and inward, and a curve of the right leg, with its concavity forward and outward, across which concavity the left leg lay. These unusual curves were only satisfactorily explained by replacing the child in its fœtal posture.

The author considers the case the most conclusive yet published from the point of view of the curves being due to intra-uterine pressure. Similar cases have been reported by Barlow and by Makin.

In discussing this case ASHBY referred to the case of an infant brought to the hospital when fourteen days old with five fractures of the long bones. There was also marked craniotabes and typical deformed rickety chest. When seen nine months after, the infant had two teeth and there was no sign of rickets. Excluding cases of achondroplasia, there was a fair number of cases recorded of so-called intra-uterine rickets; but, as far as he knew, very few had been examined pathologically shortly after birth. He had some doubts as to whether such cases were true rickets. He thought possibly they were really cases of osteoporosis, the result of malnutrition during intra-uterine life. They resembled the cases of osteoporosis found in puppies fed on food in which there was a deficient amount of lime-salts.

Symptoms of Typhoid Fever in Infancy and Childhood.—J. P. CROZER GRIFFITH (*Journal of the American Medical Association*, 1901, vol. xxxvii., No. 7) very succinctly sums up the symptomatology of typhoid fever in young patients in the following sentences:

Most cases of this disease, when affecting the very young, are of the ambulatory type. The child is hardly indisposed, although anorexia and headache are sometimes noted. In a minority of patients vomiting may usher in the disease and fever may be high at the outset.

The mortality of typhoid in childhood is not far from 3 per cent., and, roughly speaking, the younger the child the better the prognosis. Thus the mortality is less in the first than in the second quinquennium. As in adults, the roseola is commonly but not universally present. In some cases the rash is very abundant, covering the whole integument.

Enlargement of the spleen is doubtless constantly present, although not always discoverable. Epistaxis is often present, although exact statistics are wanting. The course of the disease is distinctly shorter in the young, and the average duration is about seventeen days.

Diarrhœa is much less frequently present in the child, while, on the other hand, vomiting, rare in the adult, is of common occurrence, not only as an initial phenomenon, but throughout the disease, and it has even been seen as a terminal symptom in cases ending fatally. Tympanites and hemorrhage are of rare occurrence in the child, and the same may be said of perforation. The nervous phenomena are not marked in childhood.

Finally, as a point of great practical significance, it should be stated that the onset of typhoid in the child may simulate meningitis to a more characteristic extent than in the adult.

Observations upon Milk Coagulation and Digestion.—FRANKLIN W. WHITE (*Journal of the Boston Society of Medical Science*, 1901) has made some important observations as to the value of various methods of rendering the proteids of cow's milk more readily digestible. The scope of his inquiry covered the following questions: First, whether or not cereal decoctions render the milk curd more fine and soft than a simple dilution with water; second, whether one cereal is better than another for this purpose; third,

whether or not the property of yielding a fine curd is wholly due to the starch in solution, and, if so, what per cent. of starch is desirable; fourth, what is the value of certain modifications of the method; and fifth, what is the effect of lime-water and albumin-water upon the casein curd.

The conclusions of his study are summarized as follows:

1. Dilution of milk with cereal decoctions of proper strength renders the casein curd much more fine, soft, and digestible than simple dilution with water. There is no difference in the action of various cereals, such as barley, oats, rice, and wheat.

2. The above property is due mainly, if not wholly, to the starch in solution. The most desirable amount of starch in the milk mixture for practical use is approximately ¾ per cent.

3. Diastase, by converting the starch into dextrine and maltose, promptly lessens and removes the action of cereal waters upon casein. Its addition, therefore, is not a practical measure when the action upon the curd is desired.

4. Albumin-water has no practical value as a diluent of milk.

5. Lime-water added to milk has no more effect than water upon the character of the curd produced in the animal stomach.

The Pathogeny of Night-terrors.—Rey (*Revue mensuelle des Maladies de l'Enfance*, May, 1901, p. 217) reaffirms the conclusions of his earlier studies on this subject (*Jahrbuch für Kinderheilkunde*, N. F., Bd. xlv.). The views of various authorities as to the etiology of night-terrors in children are by no means in accord. Some consider these attacks as a symptom of another disease, as of an intestinal affection, for example; others believe that they constitute a disease sui generis, an idiopathic affection of the brain; while yet a third class hold that there are two forms, one primitive, another secondary. Rey maintains that the phenomenon is developed and is always determined by an obstacle to respiration and hæmatosis, which is either direct or of reflex origin. Both forms are simply the effect of a slow and prolonged intoxication by carbonic acid. In the attack all the symptoms of a slow poisoning by carbonic acid are reproduced—suffocation, loss of consciousness, with hallucinations, followed by amnesia after recovery, and enfeeblement of muscular motility. The slow onset of this intoxication explains why the attacks never commence until three or four hours after the child has gone to sleep. Another state which results from the same cause, but exhibits symptoms of less intensity, is the nightmare of adults. The child does not raise himself quickly, but in a maladroit fashion, and cries without being able to articulate intelligible words; the tongue is rigid, and its movements are made with difficulty; when taken up in the mother's arms the child's limbs seem heavy and are moved very slowly against the body. All these signs are the result of a paresis of the muscles. As soon as he can speak he seems oblivious of what is passing on about him; he does not speak of anything but his hallucinations, and after a certain time which suffices to supply the want of oxygen and to purify the blood of its surcharge of carbonic acid, he asks simply to be put back in bed to continue his interrupted slumber.

According to the author's observation the susceptibility to night-terrors is not increased by the nervosity of the child; but the tendency seems to be

in almost direct proportion to the intensity of obstruction to nasal respiration. Children with adenoid vegetations are almost always restless during sleep. They turn and twist constantly during the night, and very often suffer from night-terrors. The same condition occurs when nasal respiration is interfered with by other obstacles, such as a tumor, a foreign body, or even a simple rhinitis. A confirmatory circumstance is that adenoid vegetations are particularly frequent at an age at which night-terrors are most frequent—that is, from three to ten years. From ten years to fifteen years both diminish in frequency. Moreover, both nocturnal pavor and adenoid growths are more frequently encountered in girls than in boys.

Carbonic-acid intoxication also produces the fright and hallucinations observed with children during the fitful slumber of an attack of pneumonia, pleurisy, angina, or bronchitis. In children who are subject to night-terrors even the slightest obstruction to nasal breathing suffices to precipitate an attack, a coryza or the mere presence of crusts obstructing the already narrowed passages being sufficient to cause it.

In the cases produced by intestinal disturbance, an overloaded stomach, or intestinal worms, it is difficult at first sight to explain a carbonic-acid intoxication. But here it suffices to remember that even according to Silbermann, who considers the affection as a disease sui generis, symptomatic night-terrors would be a reflex neurosis of the termination of the pulmonic vagus starting from the gastric terminals, a reflex which produces dyspnœa and sensations of suffocation. Both forms of the affection, therefore, result from the same cause, and there is no ground for distinguishing an idiopathic and a symptomatic form, since both are symptomatic of a carbonic-acid intoxication, direct in one case, indirect by reflex action in the other.

THERAPEUTICS.

UNDER THE CHARGE OF

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Hydrophobia and the Pasteur Methods.—DR. C. W. DULLES speaks of the early trumpeting of the Pasteur method advocates and the development of institutes for the treatment of the disease. He makes a strong and logical arraignment of the early figures, showing that they were founded on the most flimsy sort of grounds, and from an analysis of recent statistics shows that the mortality from hydrophobia in France, with its institute, is thirty times as great as that in Germany, without such treatment methods, the ratio of affected dogs being, of course, included in this comparison. In

France the number of deaths is stated to be greater after the employment of the Pasteur methods than before, the number of affected dogs being considered. He criticises the report of Salmon, of the United States Government, for adopting as facts faulty statistics, and basing upon them conclusions which will only mislead and cause unnecessary death. The author advocates a dog-muzzling law, the use of silver nitrate as a caustic, and the employment of narcotics and restraint in the attack.—*Medical Record*, 1901, vol. lx., p. 41.

Massage in Sciatica.—Dr. R. Judica advocates the abandonment of all ointments, pills, embrocations, opiates, etc., in the treatment of this condition, and recommends almost unqualifiedly the use of persistent massage. This may follow one of three methods, or all three may be employed. He mentions: 1. Schreider's method, which aims to render the limb temporarily bloodless, at the same time stretching the sciatic nerve by both active and passive means. These procedures are considered not advisable. 2. Simple massage by slow, passive movements, which, as the pain may permit, become more extensive and complete. A rest of a few moments is allowed between each movement. 3. Negro's method, which is strongly recommended. In this the patient is made to lie upon his stomach and face, the gluteal muscles being distinctly relaxed. The thumb is placed on the sciatic notch, and, with the other thumb superimposed, strong pressure is brought to bear from fifteen to twenty seconds on the nerve. Some slightly lateral and rotatory movement of the thumb is permitted during this time, but it should not be raised. This treatment is repeated after five minutes. If this is carried out on alternate days for six or seven times good results are to be expected. The author has had excellent success even in long-standing and chronic cases.—*Gazetti degli Ospedale*, 1901, vol. xxii., p. 860.

Neuritis Treated by Currents of High Frequency.—Drs. A. Denoyès and H. Lagriffoul report on the treatment of an arsenical and a post-influenzal neuritis by D'Arsonval's high-frequency currents. The results were very satisfactory.—*Archives d'Electricité medicales*, 1901, No. 7, p. 281.

Treatment of Chronic Hydrocephalus.—Drs. William Ewart and W. Lee Dickinson advocate the process of "artificial pneumocephalus" for the treatment of this condition as suggestive of what might be done for it. The method is worthy of recording. They tap the ventricles with a fine trocar; the intracranial pressure is relieved by the escape of a certain amount of fluid, and they substitute for it aseptic air in such a manner as to avoid the symptoms so commonly noted after complete removal of fluid. Their provisional conclusions are of interest: 1. With due precautions the fluid of chronic hydrocephalus may be completely evacuated from the yet unclosed skull of infants, and aseptic air may be allowed to take its place. This operation may be repeated without detriment and with scarcely more risk than belongs to the usual method of paracentesis. 2. In unfavorable cases of moderate effusion a single operation may suffice. Continual oozing from the puncture for a few days after the removal of the tube is not unfavorable. 3. In cases of considerable effusion an obvious

indication is to relieve the brain from the weight and from the pressure of the fluid. The evacuation is facilitated by the introduction of aseptic air. By a timely repetition of the operation a hydrocephalic infant might be enabled to carry the weight of the head, and, if the treatment were begun sufficiently early, permanent damage to the brain tissue might be avoided and a normal development might ensue. 4. In large heads while hydropneumocephalus persists a considerable splashing sound is readily obtained. There is obvious risk in eliciting this sound by forcible succusion, and for the same reason any abrupt movement of the head should be avoided.—*British Medical Journal*, 1901, No. 2123, p. 602.

Intramuscular Injections of Calomel in Gonorrhœal Arthritis.—Dr. M. Thomas, adopting the method advocated by Grosse, has treated a number of patients suffering from gonorrhœal arthritis by intramuscular injections of calomel. Doses of half a grain were injected at intervals of from two to four days. The results are reported as specific, but one patient had a return of his arthritis and another developed stomatitis.—*Le Mois Thérapeutique*, 1901, vol. ii., p. 59.

Value of Phosphorus Therapy in Rhachitis.—Dr. A. Monti says that the recent discussions concerning the use of phosphorus in rhachitis contain no new points of view. Both Hahnemann and Trousseau recommended, and later rejected it. He is of the opinion that this drug is of little or no value in such condition, and maintains that the early experimental studies were not free from error, and that the results obtained in the hands of clinicians are unsatisfactory and open to doubt.—*Wiener klinische Wochenschrift*, 1901, vol. xiv., p. 180.

Sidonal as an Aid to Uric-acid Excretion.—Dr. M. G. Bardet reports on the use of this drug, re-introduced into therapeutics in 1899. It is piperazine quinate, first prepared in 1868 by Lanthemann, and employed as a therapeutic agent by Rabuteau in 1872. It decomposes in the body into quinone and then into benzoic acid, and is supposed to form hippuric acid by combining with uric acid—a chemical impossibility, according to Rabuteau. Clinically, certain facts seem to point to a certain amount of efficiency. Bardet had under observation three patients afflicted with mild gout, to whom daily doses of from forty-five to seventy-five grains were given. After a month of medication the patients had improved, and weekly examinations of the urine seemed to show a diminution in the amount of uric acid eliminated. The results were not startling enough to encourage him to proceed further.—*Le Mois Thérapeutique*, 1901, vol. ii., p. 59.

Means of Arresting Acute Endocarditis.—Dr. Richard Caton advocates three procedures for the treatment of this condition—physiological rest, stimulation of trophic centres, and the administration of sodium iodide. The rest should be absolute, the patient remaining quietly in bed for a period of at least six weeks. The head should be low; sleep is encouraged, pain prevented, and all mental excitement avoided; the diet should be light and stimulating. Salicylates, the author holds, are of more value for

the joint condition than they are for the endocardium or myocardium. They should be employed, however. A second step in the treatment is by means of small local blisters. Their effect is due entirely to the stimulation of the vasomotor and trophic nerves. He applies in succession a number of small blisters, an inch in diameter, to the wall of the chest between the clavicle and the nipple. Finally, to aid in the absorption of effused material, sodium iodide is advocated.—*British Medical Journal*, 1901, No. 2128, p. 1049.

Schneiderlin's Anæsthesia.—Dr. B. Korff reports on this author's recent contribution to the subject of a new method in anæsthesia whose object was to obtain a non-dangerous continuous anæsthetic that did not worry the patient with excessive manipulations and cumbersome apparatus, and was free from the unpleasant after-effects of vomiting, coughing, etc. Schneiderlin believed that in a mixture of hyoscine hydrobromate, one two-hundredth of a grain, and morphine sulphate, one-seventh of a grain, he had obtained this ideal combination. He advises this dosage twice hypodermically at an interval of from one to two hours. Korff has modified this method as follows: He injects one one-hundred and sixtieth of a grain of hyoscine and one-sixth of a grain of morphine twice at an interval of two hours, and then applies a few drops of chloroform in a mask until complete anæsthesia is reached. Little chloroform is therefore needed, and the anæsthesia is profound and satisfactory.—*Münchener medicinische Wochenschrift*, 1901, vol. xlviii., p. 1169.

Use and Therapeutic Indications of Iodopyrine.—Dr. Wilhelm Baum has been using this drug in the medical clinic at Halle, and recommends it highly as an alternative or adjuvant to potassium iodide when this latter is badly borne by the patient or does not seem to be effective in the usual doses. With reference to its mode of medication, the author finds it ineffectual as an ointment, but internally, in doses of three or four drachms a day or more, it is effectual. It may also be emp'oyed in hypodermic medication in doses of one and one-fourth drachms of a 25 per cent. solution. The gluteal regions are preferable for injection.—*Therapeutische Monatshefte*, 1901, vol. xv., p. 641.

Heart Disease and its Treatment.—Dr. Graham Steele considers in detail various forms of cardiac lesions, and, under the head of treatment, says: It is the muscle of the heart that can almost alone be influenced by treatment. Certain pathological events strike directly at the heart muscle, and for such treatment is of little avail. These are the toxic degenerations—chief of which is that of rheumatic infection—and mechanical conditions which cripple the supply of nourishment supplying the cardiac muscle, such being narrowing of the coronary arteries or thrombosis of the coronary branches. Alcohol degenerations, he holds, offer the best field for successful treatment. Digitalis, he says, is by far the best drug of the materia medica, but it should be among the last resources of the physician rather than the first. Familiarity breeds contempt in the heart muscle as well as in society. Simple rest in bed induces that much-desired result—profuse diuresis, with

disappearance of dropsy, evidently by diminishing the work of the organ and enabling it to recover, and so restore the balance of the circulation often much better than drugs. The influence of diet is not to be ignored. Exercise deserves a place among therapeutic agents; massage also. If the use of digitalis and drugs of this class can be delayed, by all means it should be done. Tonics and strychnine can always be given with impunity. Dropsy and an engorged liver may surely be taken as indications for digitalis and like drugs. What is necessary to remember is that one cannot go on indefinitely with digitalis. It should always be given in courses of varying duration and as widely separated as possible. A prolonged rest in the early stages of heart disease will often long postpone the evil day, but care is requisite, when the patient gets up, that the change from rest to activity be very gradual; otherwise not only may benefit be lost, but injury be inflicted. Massage, with overfeeding, may be applied during the stay in bed in some cases. The author believes that an immense amount of harm is done in heart cases by a largely carbohydrate diet. It would seem that carbohydrates promote catarrhal inflammation, and in heart disease—for this reason, among others—they should be withheld or rather greatly diminished. The congested liver of heart disease and the crippled state of the organ, whose functions are probably augmented by a largely carbohydrate diet, should not be forgotten. After a certain age, apart from heart or other disease, the customs of society should be abandoned, and a simplification of diet adopted as well as a diminution of carbohydrates made. Thus the patient is allowed toast without stint at breakfast, the bread cut thin and toasted crisp and buttered cold, weak tea or coffee sweetened with saccharin, and an egg or two. Fruit may follow. At midday fish, fowl, or red meat, with well-boiled green vegetables, form the meal in most cases, but a custard pudding or cheese and butter may be added. Not a crumb of bread or biscuit is, however, permissible, and not more than half a small tumblerful of water is to be drunk, with, in many cases, half a glass of spirits added. In the evening a similar meal is given. Between the midday and evening meals a small cup of Chinese tea, sweetened with saccharin, may be given, but no bread or biscuit is permissible. On such a diet the cardiac sufferer is most likely to thrive. If he insists on a big drink—not likely when his carbohydrates are cut short—he should have it at a time far removed from his meal-times. If the patient craves for water, it should not be given with his food. For the treatment of the gastric catarrhs promoted by the venous stasis of heart disease, there is nothing like twelve or twenty-four hours' abstinence. In milder cases peptonized milk or koumiss should constitute the only food during the attack.

With reference to drug treatment: digitalis is the great agent to be employed. The drug acts slowly; and a diuresis, the great mark of its beneficial action, is never induced by it until the lapse of a certain time. If a diuresis sets in at once on its exhibition, such diuresis is spontaneous, in all probability. Occasionally a heart that has failed to respond to digitalis will respond to strophanthus, and occasionally caffeine citrate or diuretin will succeed in establishing a diuresis after both digitalis and strophanthus have failed, by their action on the kidney rather than the heart. The relief of dyspnœa and the promotion of euthanasia by the hypodermic administration of

morphine and atropine is recommended by the author. Paroxysmal dyspnœa is the symptom that specially indicates the use of morphine, and such dyspnœa is often found to be associated with considerable arterial tension. At first nitrites and alcohol give relief, but later recourse must be had to morphine and atropine. As conditions that militate against the use of morphine, much accumulation of bronchial secretion is a stronger contraindication than kidney disease. Indeed, some cases of heart failure in Bright's disease derive great benefit from its use, and stand fairly large doses; but the first dose should always be small; one must feel one's way to the dose that brings relief without risk. One thing is essential for the practitioner who treats heart disease: it is that he should never know when he is beaten while his patient is alive. When his patient is dead he should at least have the consolation of having fought a good fight on his behalf.—*Quarterly Medical Journal*, 1901, vol. ix., p. 347.

GYNECOLOGY.

UNDER THE CHARGE OF

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ASSISTED BY

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Surgical Treatment of Cancer of the Ovary.—ESTER and PUESCH (*Revue de Gyn. et de Chir. Abdom.*, 1900, No. 6) from a study of 372 cases draw the following conclusions: 1. The immediate mortality of abdominal section for this condition is not so great as to furnish an argument against surgical intervention, being about 21.3 per cent. 2. Though recurrence of the disease occurs in 59 per cent., it is not greater than after extirpation of the cancerous uterus. The number of cases in which a permanent cure is obtained certainly justifies an operation, without which a fatal termination is inevitable. 3. While operation is entirely justifiable, it is not proper to operate in all cases. The cancerous ovary should only be removed when on opening the abdomen it is found that neighboring organs are involved, or metastatic foci are discovered, or even when the poor general condition of the patient leads to the inference that the disease has become disseminated.

Curettement and Drainage of the Uterus for Salpingitis.—BEAUSSENAT and BLUM (*Revue de Gyn. et de Chir. Abdom.*, 1900, No. 6) bring up the old question of the value of intra-uterine treatment in certain affections of the tubes, which they affirm was originally proposed by Walton in 1887. Twenty cases are reported in detail in which endometritis was associated with salpingitis. They found that catarrhal salpingitis, either single or double, was cured by curettement and prolonged drainage; thickened, distorted tubes were reduced in size, and favorable results were noted, even in

the case of old pus-tubes. Cases with acute symptoms are, of course, excluded. The *rationale* of the treatment, according to the writers, is that in consequence of the dilatation, the uterus becomes softer and the uterine opening of the tube less contracted; by curettement the hypertrophied endometrium, which is the cause of tubal disease, is removed; and by prolonged drainage the uterine canal is kept patent, so as to allow exit of the secretions from the uterus and tube.

[It is rather interesting to observe a revival of the theory of tubal drainage, which has been so strongly opposed on anatomical grounds. Without questioning the value of curettement in these cases, we have never been convinced that the operation actually reopens an occluded tube.—ED.]

Anatomy of the Round Ligament.—SELLHEIM (*Beiträge zur Geb. und Gyn.*, Band iv., Heft 2), from careful dissections in the cadaver, notes several points of practical interest in connection with Alexander's operation. He was always able to find the ligament at the external ring. Both the mass of fat (mentioned by Imlach) and the nerve are uncertain guides. Within the canal, especially at the middle third, the cord is attached to surrounding tissues by fibres of muscular and connective tissue.

The writer regards an open canal of Nuck as a rare condition. He never found it in any of his subjects. The ligaments are longer in nulliparæ and thicker in multiparæ. In the latter a process of peritoneum is often carried into the canal; less frequently the cord may be cleft. Both of these conditions result from the process of involution following childbirth.

To expose the ring he recommends an incision, two inches long, beginning about half an inch inside of the pubic spine, and insists on blunt dissection after incising the skin and superficial fascia. No attempt should be made to pick out the tissue at the external ring, for fear of dividing some of the fibres of the ligament. It is not necessary to open the canal.

Retroversion without Symptoms.—E. SCHROEDER (*Zeitschrift für Geb. und Gyn.*; Band xliii., Heft 3) examined 411 patients, with the view of determining how many with retrodisplacement presented no symptoms referable to the condition. His conclusions are that the normal position of the uterus is anteflexion. In 25 per cent. of his cases retrodisplacement was present without giving rise to any painful symptoms, retroflexion being only half as frequent as retroversion; hence the inference that retroversio flexio is a common condition requiring no treatment in itself.

Tetanus following Vaginal Fixation.—MENZER (*Zeitschrift für Geb. und Gyn*, Band xliv., Heft 3) reports a case of tetanus which developed on the eighth day after a strictly aseptic operation, with afebrile convalescence, and terminated fatally three days later. The wound presented an unhealthy discoloration and secreted a brownish pus, which, injected into mice, produced typical phenomena of tetanus in a few hours. No tetanus bacilli could be isolated. Injections of antitetanus serum and salt solution were used without benefit. The result of the autopsy was negative. There was no evidence of inflammation in or around the vaginal wound. The writer was unable to explain the source of infection unless on the theory that the

tetanus germ was present within the vagina at the time of operation and was not destroyed during the usual cleansing process. It is interesting to note that a hysterectomy was performed in the same room on the same day, the patient having a normal convalescence.

Vaginal Hysterectomy as a Radical Operation for Cancer of the Uterus.—WINTER (*Zeitschrift für Geb. und Gyn.*, Band xliii., Heft 3) after careful analyses of the statistics of different countries refers to those of the Berlin clinic as the most favorable bearing on the question of the permanent cure of uterine cancer by vaginal extirpation. Up to the year 1892, 10 per cent. of the patients operated upon were free from recurrence at the end of five years. Since that year the number of cures was 15 or 20 per cent. The writer concludes that the vaginal operation is not satisfactory as a radical method of treatment, and turns to the abdominal as possibly the method of the future, although sufficient statistics have not yet been collected to allow any positive deductions.

Fibroma of the Abdominal Wall.—PINKUSS (*Zeitschrift für Geb. und Gyn.*, Band xliv., Heft 3) reports a case of fibrous tumor of the abdominal wall, of traumatic origin, in a young woman four months pregnant. She received a blow in the abdomen, which was followed by severe local pain and the rapid development of a swelling, so that it was decided to extirpate in spite of her condition.

The writer believes that this case confirms Olshausen's statement that such fibromata, which grow rapidly during pregnancy, originate as hæmatomata, due to tearing of the deeper muscular fibres, the fibrous growths subsequently springing from the fascia.

Incipient Carcinoma of the Portio Vaginalis.—FRANQUÉ (*Zeitschrift für Geb. und Gyn.*, Band xliv., Heft 2), from his studies of the lymphatics of the cervix uteri and broad ligaments in the normal and cancerous portio, arrives at the conclusion that supravaginal amputation is the preferable operation in the early stages of the disease. This view is based on the fact that metastases in the body of the uterus only occur at a later stage, and also that in commencing cancroid the deeper lymphatics are not yet affected. Statistics show that in from 50 to 70 per cent. of these early cases, according to different observers, the pelvic glands were not affected. At the Würzburg clinic 27 per cent. of the patients were free from recurrence five years after high amputation had been performed. In ten out of thirteen radical abdominal operations for advanced carcinoma of the uterus no trace of disease could be found in the iliac glands. The writer further opposes Ries' teaching that it is imperative to remove the ovaries and tubes in every case of cancer of the cervix, and affirms that metastatic deposits are rarely or never found in operable cases. With reference to the radical abdominal operation, the writer raises the question whether the immensely greater risk incurred by the patient is offset by the greater number of patients who remain free from recurrence. According to his opinion, the favorable prognosis for the patient lies not so much in the extent of the operation as in the early diagnosis.

Paralysis of the Stomach and Intestines following Operation.—HERFF (*Zeitschrift für Geb. und Gyn.*, Band xliv., Heft 2) concludes a paper on this subject with the caution that especial care should be exercised in operating upon a patient with chronic disease of the stomach. Obstinate and prolonged vomiting after anæsthesia should lead to the suspicion of possible commencing paralysis of that organ. When such paralysis occurs the stomach should be promptly and thoroughly washed out, all nourishment by the mouth being withheld. Enemata and infusions of saline solution and nutrient injections are important. Faradization of the stomach and the knee-elbow and Trendelenburg's position are also recommended.

Operation for Prolapsus Uteri.—DÜHRSSEN (*Centralblatt für Gynäkologie*, 1901, No. 29) now adopts the following method: After amputation of the cervix a transverse incision is made through the anterior vaginal fornix, and a vertical incision is carried downward from the centre of the transverse. The vaginal flaps are dissected off laterally, exposing the bladder, the slight attachments of which to the uterus are divided and the organ is freed as high as the peritoneal fold. The uterus is anteflexed and the fundus drawn down into the wound, to which it is attached in the usual manner with three sutures of catgut or silkworm-gut, provided that the patient is not past the age of child-bearing. In older subjects it is better to remove a section of the peritoneum in order to secure a fibrous serous adhesion. After excising the vaginal flaps and closing the wound a posterior colpoperineorrhaphy is performed.

The patient is discharged at the end of two weeks, and is soon able to attend to her usual duties, the uterus remaining in normal position.

The writer replies to Gebhardt's objection to vaginofixation, especially the subsequent dystocia which may be caused, by stating that in only one case upon which he has operated has there been any complication during pregnancy and parturition. He calls attention to the most important point in his operation upon which depends the avoidance of future trouble—either not to incise the vesico-uterine fold of peritoneum at all, or, if it has been opened, to unite it carefully before tying the uterovaginal sutures.

Conservative Vaginal Compared with Abdominal Section.—DÜHRSSEN (*Proceedings of the Thirtieth Congress of the German Surgical Association*) reports 780 cases of anterior vaginal section for different conditions. He believes that most of the cases formerly treated by laparotomy can be handled equally well by the vaginal route, with a mortality less than 3 per cent. There is no external cicatrix, no danger of intestinal adhesions (?) or hernia, and the convalescence is short, most of the patients being discharged at the end of from nine to twelve days. The only subsequent complication is the possible interference with parturition, which may be avoided by suturing the peritoneum separately.

Among the cases reported were seventy ovariotomies for large cystomata, with no deaths, and twenty-eight operations for extra-uterine pregnancy, with one death from an accidental cause. Among the conservative operations performed by the vaginal route the writer mentions closure of the tube to prevent conception, salpingostomy, and ignipuncture for cystic degenera-

tion of the ovary. The latter procedure he has found an efficient way of relieving the pain and hemorrhages due to this condition.

There were seventy cases of myomectomy, in some of which the tumor exceeded in size the fœtal head at term.

OBSTETRICS.

UNDER THE CHARGE OF

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Drainage after Abdominal Section.—BURCKHARDT (*Zeitschrift für Geburtshülfe und Gynäkologie*, 1901, Band xlvi., Heft 2) reports from the clinic at Würzburg 31 cases in which, for some indication, drainage was employed after abdominal section. These cases were 6 of ovarian tumors, 18 of pyosalpinx, 2 of tubal pregnancy, 2 of suppurating uterine tumor, and 3 of peritonitis. Eight of these patients died after operation, 7 of them of peritonitis, 1 of pneumonia and pleurisy.

He concludes that drainage should be employed in the following cases: 1. When pus in large quantity has soiled the field of operation and the abdominal cavity. 2. When a large raw surface has been left by operation the walls of which are necrotic and infiltrated, and where sound tissue cannot be brought together by suture. In such a case he would employ iodoform gauze drain. 3. When a portion of a tumor must be left behind because of its firm adhesions, and especially where such adhesions are to the intestine. 4. When the bladder or intestine is injured, no matter how carefully they may be sutured. 5. When pus-tubes are so adherent that they cannot be separated from the surrounding tissue after they have been opened and emptied. In this class of cases also he would use iodoform gauze as a drain; in other cases he would drain with a glass tube.

He places very little reliance upon the microscopical examination or test by culture or puncture in cases of suppurative disease. He believes the results of such tests are too uncertain to be a safe guide in employing drainage.

The Treatment of Ectopic Gestation.—IHM, of Königsberg (*Zeitschrift für Geburtshülfe und Gynäkologie*, 1901, Band xlvi., Heft 2), reports 44 cases of ectopic gestation and the results of treatment. But 1 of these had ruptured recently. In this case the patient died soon after admission to the hospital from very extensive and uncontrollable hemorrhage. In the other cases there were 2 of suppurating retro-uterine hæmatocele, 16 of retro-uterine hæmatocele without suppuration, 19 of hæmatocele surrounding the tube, 3 of pregnancy in the latter part of gestation, 1 with intact fœtal sac at the beginning of the fifth month, 1 in which a large fœtal sac had ruptured into

the bladder, and 1 in which a retro-uterine hæmatocele had already been opened by incision through the vagina. In no case did he see an intact pregnancy with living ovum during the first month.

From the 44 cases he takes 5 which were practically beyond treatment—one of very extensive hemorrhage immediately after admission; a second had been brought from the country, with acute diffuse peritonitis caused by suppurating hæmatocele; a third, with hæmatocele surrounding the tube, declined to remain in the hospital, and left before treatment could be carried out; a fourth case had carcinoma of the uterus, and the ectopic gestation was discovered accidentally in operating for the cancer, and the fifth case was one in which, six months before admission, the vagina had been opened for retro-uterine hæmatocele. This leaves in his series 39 cases uninjured by attempts at previous treatment.

Of these 19 were treated by operation—17 by abdominal section and 2 by vaginal section; 20 were treated upon the expectant plan without operation. There was practically no mortality from operation. One patient died the day after a large suppurating hæmatocele was opened through the vagina by the thermocautery.

Ihm has collected the statistics of 12 operators, giving a mortality ranging from nothing to 25 per cent. The largest series of cases is by von Schenk—436 cases, with a mortality of 16.5 per cent. The largest number of cases without mortality is 17—that of Winter. In the 17 cases of abdominal section the writer made an effort to ascertain the subsequent history. He was enabled to investigate 15 of the 17 cases; 6 of these replied to letters and 4 reported that they were well and able to work; 1 had had two children since the operation and 2 others had slight pain upon exertion, but normal menstruation. Nine patients were examined and personally questioned; 5 said that they had no pain after leaving the hospital; 1 complained of stitch pains on the left side after rapid walking or mounting stairs; 1 suffered from pain before menstruation, 1 from pain at the time of menstruation, and 1 had developed pain in the back and in the right side. In some of the patients, examination found adhesions limiting the mobility of the pelvic organs. The tubes were thickened in some cases and the ovaries sensitive; 1 had a retroflexion of the uterus and parametritis upon the left side; 1 had a tumor the size of a hen's egg upon the left side, and adhesions behind the uterus. It was impossible to say that these changes were directly the result of the operation, or that they had not been caused by gonorrhœa or efforts made in working soon after leaving the hospital.

Of 20 cases treated without operation 13 had hæmatocele behind the uterus, and 7 hæmatocele surrounding a tube. In 4 the tumor was as large as a child's head; in 2 it filled the entire space behind the uterus; in 1 it extended two fingers beneath the navel; in 1 it extended to the anterior superior spine of the ilium; in 1 it was the breadth of a hand beneath the navel; in 1 it extended between the navel and the symphysis, and in 3 it extended to the navel. Of the cases of hæmatocele surrounding the tube, 2 had a tumor as large as a fist at the side of the womb; 2 had a tumor as large as a goose-egg at the side of the uterus, while in the others the tube was evidently enlarged. All cases were examined under an anæsthetic, and in 12 of the 20 cases there was the escape of decidual membrane from the uterus.

One of the patients, who had a hæmatocele reaching to the umbilicus, was operated upon two years before for ectopic gestation upon the right side. She had remained well after the operation.

The treatment of these cases without operation consisted of rest in bed, applications to the abdomen, regulation of the bowels, the use of stypticin; after the danger of hemorrhage had passed salt baths were given to increase absorption, and applications of iodides and glycerin or ichthyol and glycerin tampons were also made. The shortest stay in the hospital of these patients was nine days, and the longest nine weeks; 11 of these patients have since been heard from; 8 have been examined and 3 have replied to letters. One wrote that she was not as well as before; that she had great pain at menstruation and could not work hard; she had not become pregnant. A second wrote that she had irregular pains, and a third stated six months after discharge from the hospital that she could not do full work because of pain in the abdomen upon exertion. The 8 cases examined showed but partial absorption of blood, and abnormalities resulting from irritation of the peritoneum about the pelvic organs. The average stay in the hospital of patients treated without operation was five weeks. To secure the full absorption of such a hemorrhage at least six weeks must be required. The bad effects of ectopic gestation persisted for eight or nine months in patients not subjected to operation, and reducing this to the lowest possible point by allowing for other causes of ill health, it was found that patients not treated by operation suffered for 5 to 7 months from ill health after leaving the hospital. Cases subjected to operation were able to work in four weeks after leaving the hospital. The expectant treatment had no mortality, which agrees with the statistics of Winkel and Fehling, while Thorn places the mortality of patients not operated upon at 1 per cent.

When the two methods of treatment are compared it is seen that both give good results, but with an enormous difference in the time required for the cure. If the two methods of treatment are compared in the same patient, when the patient was treated by operation she speedily made a perfect recovery. When the expectant plan was pursued she was not entirely well in eight months.

Shall we, then, advise section in all cases of hæmatocele? Fehling, Küstner, and Schauta state the mortality from operation as ranging from 5 to 0.9 of 1 per cent., an average of about 1½ per cent. Thorn believes the mortality of the expectant plan to be 1 per cent. The results seem practically the same.

Ectopic Gestation with Septic Infection of the Sac.—MacNaughton-Jones (*The Lancet*, June 29, 1901) reports a case of ectopic gestation for which abdominal section was done about six weeks after the first symptoms. A tumor was present behind the uterus before the operation. In delivering the sac ruptured and some extremely fetid fluid escaped. The pelvic cavity was repeatedly cleansed with weak formalin solution and the bowel well protected from the sac, the only parts exposed being the edges of the incision. A gauze drain was left. The patient did well at first, but symptoms of septic infection developed; the abdomen was reopened, and the margins of the abdominal wound found to be sloughing. Death occurred on the seventh day after the operation.

On examining the specimen the wall of the sac was infiltrated with blood clot and fibrin, and pus was found between the inflamed tube and placental tissue. As the sac was adherent to the bowel, it is reasonable to suppose that infection occurred through the intestine.

Fibroid Tumors Complicating Pregnancy and Labor.—DONALD (*Transactions of the Obstetrical Society of London*, 1901, vol. xliii., p. 180) contributes a paper under the above title. He considers the risks which fibroids cause during pregnancy to be rapid increase in the size of the tumor, with severe pain and great distress, incarceration of the tumor in the pelvis, serious pressure on the bladder, degeneration of the tumor through diminished nutrition, excessive rotation of the pregnant uterus, and abortion or premature labor.

In these cases abortion or labor is often complicated by obstruction of the birth-canal from the tumor, by malpresentations, by retention of the placenta or membranes, or by extrusion of the tumor during labor. Fibroids render septic infection much more likely than in cases without them.

These cases may be divided into those in which pregnancy is allowed to go to term and those in which it is necessary to interfere in the earlier months. It is usually best to allow the pregnancy to go to term, and then proceed in accordance with the conditions present. Cæsarean section is usually the best method of delivery. He reports a case in which pregnancy went until nearly term, when albuminuria and rapid failure in health made delivery necessary. Cæsarean section and hysterectomy were successfully performed. In the earlier months the writer does not believe that abortion should be induced. Septic infection occurred in one case coming under observation, and the patient was severely ill, but recovered. The tumor was removed later on by abdominal section. In three cases of abdominal hysterectomy for rapidly growing fibroids in the early months of pregnancy a successful result was secured.

When the tumor is so situated that it can be removed in the early months of pregnancy, this should be done, and thus the life of the child may be saved. The removal of subperitoneal pedunculated fibroids is not usually attended with much difficulty. Sessile tumors require enucleation and stitching of the gap in the uterine wall. Should this gap be extensive, hysterectomy is necessary.

Pseudocyesis.—In *La Semaine Médicale*, July 10, 1901, RAPIN reports the case of a patient who had an abortion at six months during the first year of her marriage. After this she had a catarrhal metritis and an attack of acute peritonitis, from which she recovered.

She gradually developed enlargement of the abdomen, with many of the signs and symptoms of pregnancy, and with the firm belief that she was in that condition. She finally came into false labor, having pains for some hours with distention of the abdomen. During the time of her supposed pregnancy her menstruation continued regularly.

Facial Paralysis after Spontaneous Labor.—In the *Centralblatt für Gynäkologie*, 1901, No. 45, GRÖNE reports the case of a child born in face presen-

tation and delivered spontaneously after a tedious labor. The child's face was swollen from the mouth to the eyes. Upon examination an exostosis was found behind the symphysis, to the right of the median line, and extending parallel to the ramus of the pubes. This growth had a smooth surface, and hence had not lacerated the face of the child. The pelvis was otherwise normal in contour. The promontory of the sacrum could not be reached by internal examination. The facial paralysis was upon the left side, and disappeared spontaneously in a few weeks after labor.

OPHTHALMOLOGY.

UNDER THE CHARGE OF

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AND

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The Evolution of the Ophthalmoscope.—THEOBALD, of Baltimore (*New York Medical Journal*, June 22, 1901), in an address reviewing the history of the ophthalmoscope which has now reached the fiftieth anniversary since its invention, remarks that the problem which the investigators, with the exception of Cumming, were endeavoring to solve was: Why the pupil appears black ordinarily, while under certain unusual conditions it emits a reddish light. Prevost, of Geneva, about 1810, noticed that the luminosity of the eyes of certain animals which had long been known disappears in a perfectly dark room. Rudolphi further showed that it was necessary to look into the eye in a certain definite direction to perceive the reflex. It had also long been known that in certain pathological conditions, such as intraocular growths and detachments of the retina, the eye emits a faint light. Cumming, 1846, then a student in the London Hospital, showed how a luminous appearance or reflection might be obtained in the normal human eye by examination in a dark room with the patient ten or twelve feet from the light source, while the observer's eye looks as nearly as possible into the observed in a direct line between the light and the eye. He supposed that the reflex which could always be obtained in normal eyes would disappear under pathological conditions, and thus furnish a means of diagnosis. Brücke, in 1847, placed a tube through the flame of a candle, and thus obtained a reflex, besides employing a method similar to that of Cummings; he also related an observation of a medical friend who, wearing concave glasses, obtained a reflex from the pupil of a person standing in front of him when the light reflected from the glasses entered the observed eye. In 1849 Babbage contrived an ophthalmoscope by scraping off a little of the silvering from the back of a plane mirror which he set obliquely in a suitable tube. This instrument more nearly resembled the modern instrument than that of

Helmholtz. Babbage, who seems to have been unfortunate in nearly all of his ventures, failed to have his instrument brought before the profession and properly appreciated. His friend, Wharton Jones, published an account of it in 1854, and thus secured to Babbage the credit due him.

After according to Helmholtz the credit of inventing the parent of all later ophthalmoscopes, the first instrument with which the fundus of the living eye was distinctly seen, and mentioning him as the first to suggest the indirect method, Theobald asserts that what was essential in his ophthalmoscope was not wholly original, and what was original was not only distinctly not essential, but was the outcome of a fortunate misconception on his part. This consisted in the insertion of a concave lens between the glass plates and the eye of the observer. Helmholtz was led to employ the lens, supposing that the observed eye would be accommodated for the virtual reflected source of light, the rays would leave the cornea convergent to that point, and hence would need to be rendered parallel or divergent for the observing eye. Theobald claims that this is not the function the lens actually fulfils, that the observed eye has little inclination to employ its focusing power, but that the lens serves to overcome the involuntary focusing of the observer's eye. Beginners with the ophthalmoscope, as is well known, have trouble in controlling their accommodation when looking at a near object. The difficulties first experienced by Helmholtz were great. His words are: "It was at first so difficult to use that I doubt if I should have persevered unless I had felt that it must succeed; but in eight days I had the great joy of being the first who saw before him the living human retina." Eppens, an instrument-maker of Amsterdam, a few months after Helmholtz's publication, replaced the glass plates used by Helmholtz by a plane mirror with the silvering removed from a small space in the centre. In the meantime Rekoss, a German "mechaniker," added two revolving disks which carried a series of concave lenses. In 1852 Reute suggested a concave mirror in place of the plane mirror of Epnens. (The original papers of Brucke, Cumming, Reute, and Helmholtz, all in German, have been bound together in a small separate volume.) Ophthalmologists as such had taken no part in the scientific observations which led to the invention and improvement of the instrument up to the time of Reute; thereafter they were very active in constructing new models. Mery, nearly one hundred and fifty years before, had observed, as he accidentally held a cat under water, that the vessels of the retina and the color of the fundus could be seen. Upon this hint the so-called orthoscope was now invented.

Edward Jaeger, in 1856, was one of the first to appreciate the method of determining objectively the refraction of the eye by means of the ophthalmoscope. The late Dr. E. G. Loring, of New York, deserves the largest measure of credit for bringing the refraction ophthalmoscope to its present state of perfection. His first description was published in 1869. In 1872 Dr. O. F. Wadsworth, of Boston, suggested the use of a tilting mirror, which was subsequently adopted by Loring in a modified form. The older ophthalmologists were at first somewhat loath to admit the excellencies of the new instrument, but the latter rapidly made its way. It is curious to find certain names in connection with the early history of ophthalmoscopy, such as Mr. Spencer Wells and Dr. Christopher Johnson.

Ocular Complications of Mumps.—PÉCHIN (*Gaz. Heb. de Med. et de Chir.*, 1901, No. 45) calls attention to the ocular complications occasionally observed in mumps, and reports a case of iritis with infiltration of the cornea. A young man, aged twenty-two years, whose hereditary and personal medical history was negative, became affected with a severe attack of mumps (double parotiditis) February 17, 1897, followed March 1st by facial palsy of the right side and involvement of the gustatory sense of the right side of the tongue for its anterior two-thirds. The paralysis had disappeared by March 25th, but the parotid affection continued longer. At the end of April, nine weeks after the outbreak of the original disease, the ocular complications—iritis and keratitis—made their appearance. On May 31st he was first seen by Péchin. There was bilateral iritis with posterior synechia and a slight pericorneal zone. The right cornea was infiltrated in the form of a triangle with its apex above, near the centre ; the width of the base at the limbus equalled about a quarter of the corneal circumference. The vision of the right eye was 1/2, the left, 1 badly. The ocular disease was not accompanied by pain, photophobia, or lachrymation, unlike syphilitic or rheumatic iritis. The vision constantly diminished. By June 11th it had become: right, 1/5; left, 1/4. Atropine produced large and regular dilatation on the left side, very irregular on the right. Fundi well seen. On July 28th vision was: right, 1/4; left, 1/3. Bulbar conjunctiva less hyperæmic. On November 12th vision of right eye was 1/2, badly, with muscæ volitantes; fundus visible, nerve very hyperæmic. Left eye vision was 1 on January 10, 1898; vision right was, 1/2 badly; left 1, when treatment was discontinued.

The Micro-organisms of the Healthy and Diseased Conjunctiva, and their Etiological Importance.—SANNA (*Il Policlinico*, 1901, No. 39) arrives at the following conclusions as to the bacteriological findings in the conjunctival sac:

1. The human conjunctiva, healthy or diseased, always contains a varied bacterial population.

2. The pathogenic species are about the same in the animals ordinarily used in experiments (rabbits, dogs).

3. The etiological importance of these bacteria depends upon the physiological conditions of the conjunctiva at a given time.

4. Those bacteria which are found only in the human subject, like the gonococcus, the diplobacillus, and Weeks' bacillus, are an exception to rule 3.

5. The pseudodiphtheritic bacilli, which are ordinarily found in the human conjunctiva, have no pathogenic power and are unable to acquire such.

6. Fränkel's pneumococcus inoculated into the normal human conjunctiva from anërobic cultures always remains innocuous.

7. Cauterization of the conjunctiva of dogs with phenic acid and irritation by croton oil always excite an inflammation with bacterial accompaniment about like that found in other inflammations of the conjunctiva in man.

8. Inoculations of the healthy conjunctiva of dogs with the secretion derived from the conjunctivitis of cauterization and irritation with the substances above mentioned, have never been found to cause inflammation.

9. The micro-organisms found in such conjunctivitis are destroyed in large part by the epithelium and other phagocytic elements of the conjunctiva.

10. The conjunctival epithelium and the other cellular elements exercise a true bactericidal action upon the micro-organisms, while the tears and secretion of the conjunctiva have only a mechanical action upon them.

Glioma of the Retina.—LAGRANGE (*Journ. de Méd. de Bordeaux*, 1901, No. 19) reiterates the statement that glioma has been wrongly considered even to this day as being beyond the resources of therapeutics—a statement he first published in a work in 1890, contrary to the opinion of classic authors of all countries—which overestimates the malignancy of glioma of the retina. Instances of cures are becoming daily more numerous as proven by the works of Panas, Rochon-Duvigneaud and Wintersteiner. Although cures are no longer considered extraordinary, still they are sufficiently exceptional. He reports four additional cases, ranging in age from ten months to nine years. These were all operated upon, and remained well at the time of this report, February 4, 1901, two years in one case; a year and a half in the second; ten months in a third; one month in a fourth.

Sulphate of Copper in Affections of the Cornea and Lids other than Trachoma.—CLAIBORNE, New York (*Medical Record*, July 27, 1901), advocates this agent in the following conditions:

1. In all acute attacks of inflammation of the cornea in which there is thickening, with a succulent, velvety appearance of the upper lid.
2. In all recurrent attacks of superficial keratitis in which the same condition of the upper lid prevails.
3. In infiltrations of the cornea which are the result of preceding inflammations, associated with the same condition of the upper lid.
4. In maculæ of the cornea in children and adults which have occurred a reasonable time after an inflammation, whether the upper lid presents the characteristic appearance or not.
5. In chronic conjunctivitis attended by thickening of the lid associated with blepharitis.
6. In chronic dacryocystitis (particularly in those cases in which the canaliculus has been slit) attended by chronic conjunctivitis.

He applies the solid stick ground in the form of a parallelogram underneath the upper lid between it and the eyeball, so as to reach that part of the lid next to the ball, as well as to the cul-de-sac of the lower lid. The surplus blue stone is washed off with water and removed from the lower lid by a cotton-wrapped probe.

Pathogenesis of Choked Disk.—MERZ (*Archives of Ophthalmology*, July, 1901) draws the following conclusions, among others, from experiments upon dogs and rabbits: Increased intracranial tension alone is sufficient to cause choked disk. It is only necessary that the tension should be maintained uninterruptedly for a certain time. The tension need be but slightly increased—8 to 15 mm. of mercury or less. A transient increase of tension, even though often repeated, leads only to venous hyperæmia and arterial anæmia. The first clinical sign of increased intracranial tension seems to affect the retinal circulation—dilatation of the veins and constriction of the arteries. The nearer to the eye the vessels perforate the sheath of the nerve the sooner do the retinal changes appear.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF
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Supravascular Pericardial Nodules.—KNOX, in a paper entitled "Supra-arterial Epicardial Fibroid Nodules" (*Journal of Experimental Medicine*, 1899, vol. iv., p. 245), describes small nodules which are frequently seen along the branches of the coronary arteries. These he regards as differing from the *periarteritis nodosa* first described by Kussmaul and Maier, and as having no relation to endarteritis. He finds that the outer elastic lamella of the artery immediately beneath the nodule is frequently reduced or has disappeared, and considers this due to irregularities and increase of blood-pressure affecting that part of the bloodvessel wall unsupported by myocardial tissue. In consequence of this the artery wall would here tend to bulge were this tendency not restrained. The formation of dense supra-arterial nodules over the weakened area holds this tendency in check, and may therefore be regarded as an adaptive or compensatory change.

HERXHEIMER ("Ueber supravasale Pericard-Knötchen und Sehnenflecke," *Virchow's Archiv*, 1901, Band clxv., p. 248), studying the same conditions, differs from Knox in some of his conclusions. He agrees that these nodules are distinct from *periarteritis nodosa*, but does not think they depend on any primary change in the bloodvessels, since, contrary to Knox, he finds the vascular changes slight and inconstant. The external elastic lamella, to which Knox attaches special importance, Herxheimer finds to vary greatly, sometimes being increased beneath the nodules, sometimes decreased, and the same variations occur where no nodules are superimposed on the bloodvessel.

Both find that the nodules develop outside the adventitia of the artery from the connective tissue lying between the endothelial and elastic layers of the epicardium—the point of origin, as shown by Meyer and Ribbert, of the milk patches so often found on the epicardium. In all of Herxheimer's cases milk patches also occur, and their mode of origin is the same as that of the nodules. Further, in addition to definite nodules, he finds along the course of the arteries areas of simple thickening of the epicardium, and these can be traced from their earliest beginnings in the epicardial surface. Finally, he describes transitions between these processes. The simple epicardial thickenings may extend along a vessel for some distance, and in some of these places nodules occur. Nodules occur independently of vessels, and by studying serial sections the thickenings along the course of vessels are sometimes seen to be continuous with large epicardial thickenings independent of vessels. From this he concludes that the supravascular nodules and milk patches are structurally and in origin identical.

Three theories have been adduced for the origin of milk patches: (1) They originate from a circumscribed pericarditis; (2) they have a mechanical explanation in chronic vascular pressure changes; (3) they arise from

developmental irregularities, perhaps in the endothelium. The weight of evidence is against the first. The third theory is urged from the frequent occurrence of splits lined by endothelium, but Herxheimer considers these as secondary changes arising from endothelial cells caught in from the endothelium of the epicardium, and points out the infrequency of the patches in the newborn as being against a developmental origin. The second theory he supports as explaining both the patches and nodules. Both occur most frequently on the left ventricle and in cases where the evidence points to high and irregular blood-pressure. The arteries, filled with blood, project above the epicardial level and so are more exposed to friction. The nodules occur oftenest in the region of a bifurcation, where pressure is highest. They do not occur in Herxheimer's cases on the arteries lying protected in the sulcus coronarius.—H. A. C.

Bacteriology of the Gall-bladder.—MIECZKOWSKI ("Zur Bakteriologie des Gallen-blaseninhaltes unter normalen Bedingungen und bei der Cholelithiasis," *Mitt. a. d. Grenzgeb. d. Med. u. Chir.*, 1900, Band. vi., p. 307) made a bacteriological study of the bile obtained from fifteen patients on whom laparotomy was performed for diseased conditions affecting other parts than the bile passages and in whom there was no evidence of a pathological condition of the gall-bladder or ducts. He finds the bile aspirated under these conditions to be sterile in every case, and concludes that human bile obtained from a normal gall-bladder is sterile. The same he finds to be true for the gall-bladder and hepatic and cystic ducts of animals, while in the common duct organisms are usually present. The organisms often found in human bile at autopsy he regards as post-mortem invaders.

Though the bile is normally sterile, he shows that it is nevertheless a medium in which bacteria can richly multiply, though not so energetically as in bouillon. This is especially true for B. coli communis.

Mieczkowski considers mechanical conditions to be of more importance in explaining the sterility of the gall-bladder than any bactericidal power of the gall. Bacteria are swept away by the stream of bile; but if this outflow is hindered, as in cases of cholelithiasis, infection takes place. Of twenty-three cases of cholelithiasis the bile in eighteen was infected, fifteen times with B. coli communis alone and three times with this organism plus one of the pyogenic cocci. From this he concludes that cases of cholelithiasis should be regarded as infected cases, since the gall-bladder is rarely sterile, and the absolute virulence of the organisms is heightened by the difficulty in absorption of the bile escaping at the time of operation into the cavity of the abdomen.

EHRET and STALZ ("Experimentale Beiträge zur Lehre v. der Cholelithiasis," *Ibid.*, Band vi., p. 350) do not agree with Mieczkowski in regarding the bile as absolutely sterile, since they claim to be able to demonstrate organisms in the bile of guinea-pigs, dogs, and cattle, provided larger quantities (1 to 4 c.c.) of bile are used in making the cultures. The organisms, however, are few in number, since they, as well as Mieczkowski and others, obtain no growths when small quantities of bile are used.

These two observers consider that the organisms reach the gall-bladder from the intestine by way of the gall-ducts. For the proof of this they

adduce the following: If the cystic duct or common duct is ligated, and the bile allowed to escape into the peritoneal cavity, no peritonitis results, while the reverse is true when the common or hepatic duct is ligated; the last procedure interrupts the stream of bile, and so affords an opportunity for organisms to extend up the duct. In the common duct, close above the papilla biliaria, organisms are generally present in considerable numbers.

MIYAKI ("Zur experimentellen Erzeugung d. Gallensteine m. besond. Berücksichtigung d. bakt. Verhaltens d. Gallenwege," *Ibid.*, Band vi., p. 479), in an examination of the bile of seventy-six dogs and rabbits, finds the gall-bladder sterile with one exception (small quantities of bile used in making cultures). From a study of a small number of animals he concludes that in the lower part of the common duct organisms are frequently present, and these are the normal inhabitants of the intestine.

Miyaki also investigated the pathological changes in the gall-bladder necessary to bring about the development of bacteria there. Ligature of the cystic duct produces no change, while ligation of the common duct close to the papilla bilaria causes an infection of the gall-bladder. The introduction of sterile foreign bodies into the gall-bladder produced infection in three out of eight experiments, while in three more animals in which the cystic duct was also ligated the gall-bladder remained sterile. Direct injury to the gall-bladder mucosa caused no infection. Organisms introduced into the gall-bladder, with or without the production of gall stasis, can be recovered in pure culture after periods varying from three days to one year and eight days.

This work seems to show: 1. That the bile is normally sterile in the gall-bladder and cystic and hepatic ducts. The common duct, except in the lower part, is also sterile. If organisms are normally present it is only in very scant numbers. 2. That organisms will multiply in the bile. 3. That conditions favorable to their entrance are not readily produced experimentally, but when introduced the organisms persist. 4. That gallstones favor their entrance, and in cases of cholelithiasis the gall-bladder is almost always infected. 5. That the mode of infection in these cases seems to be by way of the gall ducts from the intestine.—H. A. C.

The Source and Significance of the Cell Masses Occurring in the So-called Nævi of the Skin.—ABESSER (*Virchow's Archiv*, 1901, vol. clxvi., p. 40) says the characteristic feature of the nævus is composed of epithelial-like cells which occur singly, in clumps, and in bands in the papillæ and cutis of the skin. Two views obtain in regard to their origin: The first is that they are derived from the epiderm (Durante, 1871; Unna, 1893); the second, that they arise from the mesoderm (Demiéville, from the endothelium and perithelium of bloodvessels; v. Recklinghausen, from the endothelium of lymph vessels; Ribbert, from the chromatophores). Soldan (1899) regards nævi as neurofibromata. The epithelial origin was favored in 1897 by Waldeyer and Kölliker; later by Marchand and Orth.

Abesser studied in serial sections sixteen small nævi from adults. He found three types of cells present. The most common, which he calls the typical nævus cell, is a round or oval cell of the size of those in the epidermis. It has a vesicular, epithelial-like nucleus, and lightly staining, often invisible, protoplasm. These cells occur usually in round balls in the

papillæ and the upper part of the cutis. The second kind of cell is small and flat, with a long oval nucleus, which is surrounded by a very little protoplasm; it occurs in thick bands of closely packed cells, which extend to the epidermis and deep into the cutis. The third form, called chromatophore, contains pigment; it varies greatly in size and shape; it may be branching, star-shaped, oval, or round.

The first noticeable characteristic of nævi is that a hypertrophy of the epidermis occurs in the region affected; the epidermis is thickened, the papillæ narrowed; the second characteristic is that the cells in the lowest layer of the epidermis undergo a metaplasia into nævus cells. A certain liquefaction first takes place, which separates the cells more or less from each other; the cells also lose their protoplasmic fibrillation. The cells are now free to escape into the underlying cutis. The writer compares the picture presented by the frayed epidermis between the papillæ as the nævus cells escape from it into the cutis to a splintered broom. Unna is inclined to believe that elastic and connective tissues play some part in forcing the nævus cells free from the epidermis. Frequently clumps of nævus cells are found within the epidermis surrounded by a single flattened layer of basal epithelium. Ribbert thinks that the nævus cells develop in the cutis and extend to and even press into the epidermis, thus forming the clumps of cells above mentioned.

The second, small, form of nævus cell is probably derived from the first, because all transitions in size between the two can be found.

The third form—the branched and polymorphous chromatophore—arises from the pigmented cells of the epidermis by the same process of metaplasia. This is shown by finding similarly branching pigmented cells in the epidermis and by tracing their escape into the cutis.

All three kinds of nævus cells are derived, therefore, in the writer's opinion, by metaplasia from the cells of the epidermis. They lose their protoplasmic fibrillation, but otherwise retain their epithelial character in that they produce no intercellular substance. The tumors which they give rise to should be classed as nævo-carcinomata.

[The fallacy in this work, if there is one, lies in drawing conclusions from the study of nævi from adults in whom it must be supposed that the process of development in the nævi has been quiescent for a great many years; therefore it is exceedingly doubtful if the lesions are in a stage in which they can be rightly interpreted. Nævi should be studied in young children at the time when the lesions are developing; then the process of metaplasia can be followed. The conclusions claimed as proved are too important to be accepted without further study of more favorable tissues.—F. B. M.]

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